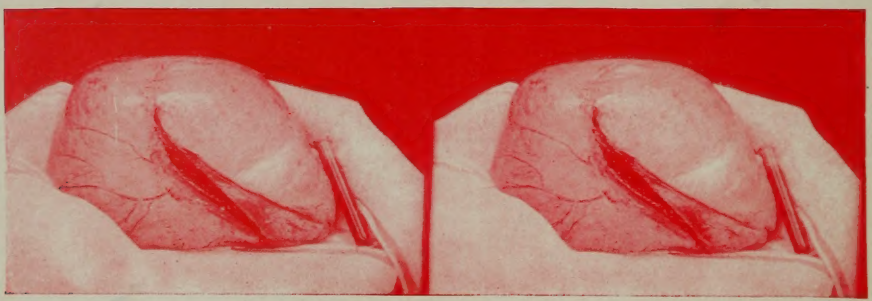


KROMOGRAM OF AN OVARIAN CYST.

The stereoscopic picture at the bottom of the page represents the combined printing of the blue, red, and yellow blocks. (To illustrate article on "The Kromskop.")

INTERNATIONAL CLINICS

A QUARTERLY

OF

CLINICAL LECTURES AND ESPECIALLY PREPARED ARTICLES

ON

MEDICINE, NEUROLOGY, SURGERY, THERAPEUTICS, OBSTETRICS, PÆDIATRICS, PATHOLOGY, DERMATOLOGY, DISEASES OF THE EYE, EAR, NOSE, AND THROAT, AND OTHER TOPICS OF INTEREST TO STUDENTS AND PRACTITIONERS

BY LEADING MEMBERS OF THE MEDICAL PROFESSION THROUGHOUT THE WORLD

EDITED BY

HENRY W. CATTELL, A.M., M.D., PHILADELPHIA, U. S. A.

Director of the Ayer Clinical Laboratory of the Pennsylvania Hospital

WITH THE COLLABORATION OF

JOHN ASHHURST, JR., M.D., LL.D., AND CHARLES H. REED, M.D.

OF PHILADELPHIA

JAMES T. WHITTAKER, M.D., LL.D.

OF CINCINNATI

WITH REGULAR CORRESPONDENTS IN MONTREAL, LONDON, PARIS, LEIPSIC, AND VIENNA

VOLUME II. TENTH SERIES, 1900

PHILADELPHIA

J. B. LIPPINCOTT COMPANY

1900

PRINTED BY J. B. LIPPINCOTT COMPANY, PHILADELPHIA, U.S.A.

CONTRIBUTORS TO VOLUME II.

(TENTH SERIES.)

ALDRICH, CHARLES J., M.D., Lecturer on Clinical Neurology and Anatomy of the Nervous System in the College of Physicians and Surgeons of Cleveland, Ohio; Visiting Physician and Neurologist to the Cleveland General Hospital and to the Cleveland City Hospital.

ALLEN, CHARLES WARRENNE, M.D., Adjunct Professor of Skin Diseases in the New York Post-Graduate Medical School.

ANDREWS, EDMUND, M.D., LL.D., Professor of Clinical Surgery in the Northwestern University, Chicago.

BABCOCK, ROBERT H., A.M., M.D., Professor of Clinical Medicine and Diseases of the Chest, College of Physicians and Surgeons, Chicago.

BALLANTYNE, J. W., M.D., F.R.C.P.E., F.R.S. (Edin.), Lecturer on Antenatal Pathology and Teratology in the University of Edinburgh; Honorary Fellow of the American Association of Obstetricians and Gynæcologists.

BARIE, ERNEST, M.D., Assistant Professor in the Paris Faculty of Medicine.

BENEDICT, A. L., A.M., M.D., Professor of Physiology and Digestive Diseases, Dental Department of the University of Buffalo; Special Consultant in Medicine, Buffalo Hospital of the Sisters of Charity.

BISHOP, LOUIS FAUGÈRES, A.M., M.D., Assistant Attending Physician to the French Hospital; Attending Physician to the Colored Hospital; Late Chairman of the Section on Practice of Medicine, New York Academy of Medicine.

BISHOP, SETH SCOTT, B.S., M.D., LL.D., of Chicago.

BISS, HUBERT E. J., M.A., M.B. (Cantab.), D.P.H., Assistant Medical Officer to the Grover Fever Hospital (Metropolitan Asylums Board).

BOOTH, J. MACKENZIE, A.M., M.D., Lecturer on Clinical Surgery in the Children's Royal Infirmary, Aberdeen, Scotland.

CATTELL, HENRY W., A.M., M.D., Director of the Ayer Clinical Laboratory of the Pennsylvania Hospital, Philadelphia.

DIEULAFOY, G., M.D., Clinical Professor in the Paris Faculty of Medicine.

ESHNER, AUGUSTUS A., M.D., Professor of Clinical Medicine in the Philadelphia Polyclinic; Physician to the Philadelphia Hospital; Assistant Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases.

GOFFE, J. RIDDLE, M.D., Professor of Gynæcology in the New York Polyclinic; Visiting Gynæcologist to the New York City Hospital.

GREEN, FRANK K., Ph.G., M.D., of Louisville.

HALL, J. N., M.D., Professor of Medicine in Gross Medical College; Visiting Physician to the Arapahoe County Hospital and to St. Anthony's Hospital, Denver, Colorado.

HINSDALE, GUY, M.D., of Philadelphia.

HOTZ, F. C., M.D., Professor of Ophthalmology and Otology in the Rush Medical School, Chicago.

KATZENBACH, WILLIAM H., M.D., Professor of Diseases of the Chest and Internal Medicine in the New York Polyclinic Hospital and Medical School for Graduates; Vice-President of the New York Academy of Medicine.

MAPES, C. C., M.D., of Louisville.

MORRIS, ROBERT T., M.D., Visiting Surgeon to the Post-Graduate and Ithaca City Hospitals; Member of the American Medical Association, of the American Academy of Medicine, and of the State Medical Society, and one of the Alumni of Bellevue Hospital.

MURPHY, JOHN B., M.D., Professor of Clinical Surgery in the College of Physicians and Surgeons, Chicago.

OLIVER, CHARLES A., A.M., M.D., Attending Surgeon to Wills Eye Hospital; Ophthalmic Surgeon to the Philadelphia Hospital, Philadelphia.

PATRICK, HUGH T., M.D., Professor of Neurology in the Chicago Policlinic; Associate Professor of Nervous Diseases in the Northwestern University Medical School; Neurologist to the German Passavant and Maurice Porter Hospitals.

ROBISON, JOHN A., A.M., M.D., Assistant Professor of Medicine at the Rush Medical College; President of the Chicago Society of Internal Medicine; Attending Physician to the Presbyterian Hospital.

SENATOR, H., Director of the Third Medical Clinic and Professor of Special Pathology and Therapy in the University of Berlin.

SKENE, ALEXANDER J. C., M.D., of Brooklyn, N. Y.

SMITH, ANDREW H., M.D., of New York.

SOUCHON, EDMOND, M.D., Professor of Anatomy and Clinical Surgery in the Tulane University, New Orleans, Louisiana; Fellow of the American Surgical Association.

STRICKER, LOUIS, M.D., of Cincinnati, Ohio.

THOMPSON, JOHN A., of Cincinnati, Ohio.

TODD, FRANK C., M.D., Professor of Clinical Ophthalmology and Otology in the University of Minnesota, Minneapolis.

VALUDE, E., Physician to the Clinique Nationale des Quinze-Vingts.

WALSH, JAMES J., M.D., Instructor in Diseases of the Chest and Internal Medicine at the New York Polyclinic Hospital and Medical School for Graduates.

WALTON, G. L., M.D., Clinical Instructor in Neurology in Harvard University; Physician to the Neurological Department of the Massachusetts General Hospital.

WILSON, J. C., M.D., Attending Physician to the Pennsylvania Hospital, Philadelphia.

CONTENTS OF VOLUME II.

(TENTH SERIES.)

NEW INVENTIONS.

THERAPEUTICS.

MEDICINE.

NEUROLOGY.

SURGERY.

OBSTETRICS AND GYNÆCOLOGY.

DISEASES OF THE EYE AND EAR.

DERMATOLOGY.

AMERICAN MEDICAL ASSOCIATION.

LIST OF ILLUSTRATIONS TO VOLUME II.

(TENTH SERIES.)

PLATES.

FIGURES.

New Inventions

THE KROMSKOP.

AMPLIFICATION OF REMARKS MADE BEFORE THE PHILADELPHIA COUNTY MEDICAL SOCIETY, MAY 23, 1900.

BY HENRY W. CATTELL, A.M., M.D.,

Director of the Ayer Clinical Laboratory of the Pennsylvania Hospital, Philadelphia.

Ever since the discovery of Daguerre that a permanent record of an image could be made by what we now call photography, earnest investigators have endeavored to reproduce in a picture not only the lights and shades of the image, but its proper colors also. Indeed, the early workers were confident that their hopes in this direction would be realized within a short time. I have been told on reliable authority that in the forties the then professor of chemistry in Princeton College, while experimenting with the making of daguerreotypes, actually reproduced the natural colors in a picture. And so ignorant was he of the true nature of the daguerreotype that it was thought, both by him and by his assistant, my informant, that a colored photograph might be reduced by following out the details of the process itself. Much to his disappointment, however, the picture faded away, and the professor was never able to secure a like result, though he repeated his experiments many times with similar solutions in the hope of so doing. It is possible that he may have hit upon the interferential method of Lippmann for producing colored pictures, which consists in passing the objective rays through the photographic plate and then reflecting them back by a mercury trough.

In 1861 Professor James Clerk Maxwell suggested that three separate photographs could be made through solutions of the three fundamental colors, and that the same colors properly placed before

the three magic lantern slides might blend them together so as to produce the color-values of the object photographed. Collen, Ransonnet, du Hauron, and others were unable to obtain practical results of any value, though careful workers in this field of research.

To Frederic E. Ives is due the credit of rendering previous theories practical by new applications of certain facts already known. His first publication upon this subject appeared in the *Journal of the Franklin Institute,* of Philadelphia, January, 1889, page 58, his first practical demonstration having been given before this Society in February of the year previous.

By taking three negatives through three *composite* screens, represented by the colors red, blue, and green, adjusted to the requirements of the negative plate employed, and, by the aid of a specially constructed magic lantern, again passing the three images through *pure* color-screens of red, green, and blue, he superposed the three pictures the one upon the other so as more or less perfectly to reproduce the actual appearance of the objects photographed.[1]

Working along this line, he devised an instrument which has received the by no means euphonious name of kromskop, which means to see color. By the use of this optical instrument one is able under suitable conditions to see a stereoscopic picture reproduced in colors. Theoretically it should be possible to reproduce every color, but practically it is found that certain colors are capable of more exact reproduction than others.

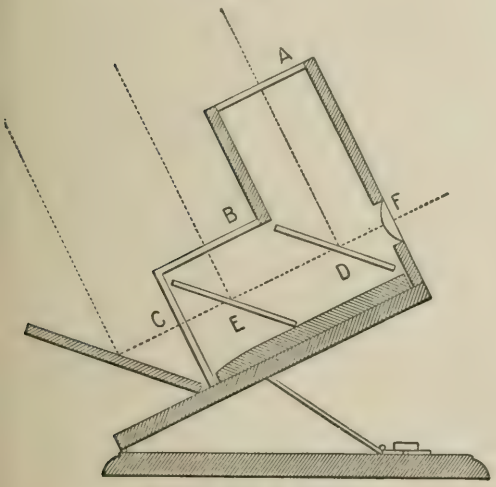
Fig. 1 shows a sectional plan of the kromskop. *A* and *B* are glasses colored respectively red and blue. *C* is an opening. Against these rest the corresponding pictures, which have been taken through composite color-screens,—red-orange, green, and blue-violet. *D* and *E* are transparent reflectors of colored glass, represented by the colors azure blue and green. The object of these colored glasses, which are complementary to the ones above, is to avoid a double reflection of the image, to correct any deficiencies in the other colored glasses, and to serve as the color-screen for viewing the green images. *F* is an opening fitted with a magnifying lens. Be-

[1] In this connection mention should also be made of the McDonough process, which follows on the work of du Hauron and Joly. There are introduced in front of the negative plate special color-screens composed of a series of fine parallel lines (about two hundred to the inch) in red, blue, and green. To secure the color-values the lantern slides are shown through screens of a similar character.

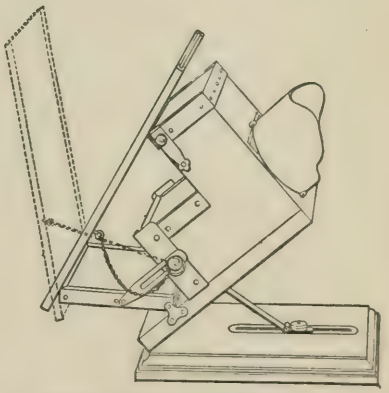
FIG. 2.—Mr. Frederic E. Ives, the inventor of the kromskop, inserting a kromogram into the instrument.

yond the open space at *C* is a reflector, which serves to illuminate the green image at *C*. The pictures at *A* and *B* are illuminated from above by direct light. The image at *C* first passes through the transparent glasses *E* and *D* and reaches the eye at the lens *F*. The blue image passing through the screen colored blue at *B* is seen by reflection from the inner surface of the glass *E*, where it is superposed upon the image of *C*, the two now appearing as one. In a similar manner the red image, passing through the red screen at *A*, is seen by reflection from the transparent mirror *D*, where it is superposed upon and becomes part of the combined images of *B* and *C*. The whole combination of the images *A*, *B*, and *C* is now taken up by the lens at *F*, where it appears as a completed image in its true colors. The glass *D* passes through the red glass and is superposed upon the images passing through the blue and green glasses.

FIG. 1.



Sectional plan of the kromskop.



Method of using ground-glass screen.

In the stereoscopic kromskop there are two lenses at *F*, which, as in the ordinary stereoscope, blend two images that have been taken from slightly different points of view into one, as in binocular vision. For this instrument there are six pictures, arranged in three double or stereoscopic sets.

By an ingenious arrangement, represented in Fig. 2, the three pairs of pictures are so held together that they naturally fall into place when put into the kromskop. This combination of pictures

is called a kromogram. When there are no pictures in place in the kromskop, the combination of glass screens should produce white. It will be found, however, that the field will have a bluish tinge, especially if there be no clouds in the sky.

The kromogram is prepared as follows: The three negatives are made with a specially constructed camera, preferably at a single exposure. The equal length of exposure is made to depend upon the size of the diaphragm, for it requires a varying period of time to take a picture through the different screens. Thus, if it needs fifteen seconds for the red, it may require six seconds for the blue and two seconds for the green. The advantage of taking the three pictures at once is manifest, as the object to be photographed is not so liable to change its shape, color, or form as when three exposures are taken. Neither are the changes of light apt to be so varied, and, if they occur, they will affect similarly all three of the pictures, and the color-effects will be alike in all. The positive record is next made by contact printing from the negative in the usual way. The glass plate is then cut in three and mounted on a special hinged frame.

In using the kromskop a good light is of the greatest importance. Other things being equal, light which is most suitable for the microscope is the kind to choose for the kromskop. The light must fall directly upon the mirror and points *A* and *B* in Fig. 1. This object can be secured by an elevation of the body of the kromskop from below or by tilting the mirror. There should be no sash-bars or other object between the sky and the kromskop. The best results which I have seen were obtained by artificial light: an arc lamp guarded by a ground-glass screen, an acetylene light, or a Welsbach burner supplemented by a weak solution of methylene blue gives excellent results. The kromskop is placed on a table and so adjusted that a whitish field will be obtained. There should be no color-fringes visible at any point.

If the sky is uneven—made up of white and blue in patches—or the light is obstructed by branches of trees or by other objects a ground-glass screen may be used as shown in Fig. 3, in which *A* represents the kromogram in place and the screen in its proper situation, while *B* represents the screen removed for the introduction of a new kromogram. The direct rays of the sun must never strike the kromskop. The desired result is obtained by the use of daylight from the north when the sky is evenly clouded.

Persons affected with color-blindness will have to regulate the instrument according to the character of their defect. Indeed, it is perfectly feasible to use this instrument for the detection of this affection, and it would be interesting to prepare a series of kromograms of the various colored worsteds used in testing for color-blindness, and then ask the patient to pick out the corresponding colors by means of kromskopic images.

Having obtained as nearly as possible a pure white field, the kromogram is now introduced, so folded that the plate containing the image which is to pass through the red glass appears on top; this plate is recognized by means of a small circular white label upon its right-hand upper corner. It is here that the kromogram is to be grasped by taking hold of the top section with the thumb and index-finger of the right hand. This brings a black card which fits in the unused space between *A* and *B* in Fig. 1. This card may be utilized for a description of the picture about to be shown. The writing or printing will therefore appear towards the operator, while the circular spot will be towards the window. The lower section—the one that is to be placed against the green glass—is now grasped at its left edge (care being taken that no finger-marks appear on the glass) with the thumb and fingers of the left hand, and lowered into the two lateral grooves which are found on each side of *C*, Fig. 1.

As soon as the first section has fallen into place, the second section is allowed to rest upon the top of the lower step. There is a slight projection just above *C*, and the joining together of the two sections of the kromogram permits an open space so that the two ribbons pass on either side.

The upper picture now naturally falls into place on the top step. Each of the plates is now shoved towards the left by a gentle tap with the fingers of the right hand. In case this does not secure exact registration, perfect adjustment may be made by means of the two screws, one on the right-hand side and the other on the left. It will be seen that by turning these screws the pictures can be thrown in or out of register; hence if an exact juxtaposition of the three images is not secured, a little experimentation will show where the error lies. As in the microscope, the use of fine adjustment causes the proper image to appear and disappear.

The eyes are now placed centrally over the lenses, as close as

possible to the shield. It is astonishing how realistic the image is which now appears. It stands out in relief, and in a good kromogram you can almost imagine that the object is before you. What a wonderful result it will be when the stereoscopic kromskopic picture shall be combined with the phonograph and the moving picture!

The kromogram is now removed by grasping it at the upper right-hand corner and elevating it. The left hand is employed to loosen the ribbons in case they become caught. The lower picture is now allowed to rest upon the table by falling flat away from the operator. The second naturally falls on top of it, and this folding is continued until the whole is in place, the round label appearing upon the outside. The kromogram should then be returned at once to a small box or put into a proper receiver.

All parts of the kromskop must be kept scrupulously clean and free from dust, in order to give the best results. Every mark or flaw in the kromogram is apt to produce a fringe of color. This can readily be seen by the introduction of an object, such as the finger, between the reflector and *C*, Fig. 1.

The reflecting mirror, the outside colored glasses, and the eyelenses may be cleaned with a silk handkerchief or a soft linen duster. This should be done with as much care as in the cleansing of a microscope. The greatest caution is necessary in cleaning the colored glass reflectors. For this purpose the springs which hold them in place are unhooked. In putting the reflectors back into place, care must be taken that the corner-scratch *X* comes in contact with a similar mark on the wooden base, otherwise the instrument will not be properly adjusted.

When the kromskop is adjusted for one kromogram, no further attention is necessary for the others; provided, of course, that the kromograms themselves have been correctly registered and have not got out of adjustment.

By means of the magic lantern kromskop, photographic pictures showing objects in their natural colors are thrown upon a screen, a use of the instrument that renders it most valuable for class demonstrations.

The lantern is so arranged that two of the three tubes—the ones upon the right and the left—can be swung aside, showing three colored pictures side by side, produced by the passage of light through the lantern slide and the proper colored glass. This is

done by the use of a lever, which, when reversed, superposes the pictures in exact register.

The uses to which these various instruments can be put are many. I shall confine my remarks, however, to the reproduction of the natural appearance of pathological specimens.

As is well known, many methods have been proposed by means of which permanent records of the appearance of fresh tissues may be preserved for future use. It was thought at one time that lithography and the wax model had solved this question, and we had in the fifties most excellent examples of this method of showing the natural appearance of the various parts of the body. No one can examine Lebert's Atlas without questioning whether we have really advanced much in the way of illustrating our medical works, or see the models of Ziegler, Talrich, and Tramond, which are to be found in most of our museums, without admiring their natural appearance. The well-known imitations of flowers in colored glass by the Blaschkis, father and son, at Harvard University, are excellent examples of the artificial reproduction of the natural colors.

A stereo-kromskopic picture is taken of the pathological specimen, such as is shown in the frontispiece, in which we see an ovarian cyst, removed by Dr. Le Conte, the fluid contents of which weighed over thirty-two pounds. The injected blood-vessels and the oviduct, with its fimbriated end, can readily be made out. To the right is a test-tube filled with some of the fluid from the cyst.

The three upper pictures represent the photographs taken through the three composite color-screens. The red negative is printed in blue, the green negative in red, and the blue negative in yellow. The lower picture represents a combination of the three colored pictures printed the one upon the other. (Frontispiece.)

In order to secure the stereoscopic effect, the lower portion of the page must be inserted into an ordinary stereoscope, when the whole will be seen to stand out in its proper relation. Had the picture been intended to illustrate the process of three-colored printing in its perfection, the plates would have been made larger and the screen-mesh smaller; but they now represent the actual size of the kromskopic picture.

In taking the pictures, orthochromatic or isochromatic plates should be employed. A photograph of the spectrum should be made, to see if the true color-values are reproduced. Next, pictures

are taken through the three screens for half-tone reproduction. Excellent results may be obtained by the use of a pyrocatechin, metol, or tolidol developer; hydrochinone should not be employed.

Pieces of tissue are then cut off for microscopic study, and the specimen is preserved by some process which will show the natural colors of the part. This is best done by the use of Kaiserling's solution, which is as follows: The specimen is placed in the position which it is to have permanently. This can readily be accomplished by proper stuffing with cotton and the use of wires and small sticks. The specimen is then laid on cotton in a jar containing formalin, 500 to 750 cubic centimetres; potassium nitrate, 10 grammes; potassium acetate, 30 grammes; distilled water, 1000 cubic centimetres.

The specimen remains in this fluid during a period of twenty-four hours, when it should be examined to see if it is thoroughly hardened; if not, it should remain in the jar until the hardening is complete. The preparation is then transferred to eighty per cent. alcohol for twelve hours and to ninety-five per cent. alcohol for two hours. The color will promptly be restored after the specimen has been placed in the alcohol. The preparation is subsequently preserved in a fluid composed of equal parts of glycerin and water, to every one thousand parts of which thirty parts by weight of potassium acetate have been added. The jars containing specimens should be kept in the dark, as sunlight causes a deterioration of their colors.

The kromskop with the kromogram is then sent to the photographic engraver. If it be thought necessary, the specimen may also be sent for comparison. In preparing the half-tones reference to the kromskopic picture should be made whenever any doubt arises. The same method is employed with the printer. He is informed that the colors must be identical with those of the kromskop, or, if any change is to be made, the required alteration can readily and specifically be indicated. If a colored drawing is to be prepared by an artist, he has before him the specimen, preserved by Kaiserling's method, showing the shape of the object, and by reference to the kromskop he can secure the proper color-values.

Therapeutics

THERAPEUTICS OF THE UNBORN INFANT.

LECTURE DELIVERED IN THE UNIVERSITY OF EDINBURGH, FEBRUARY 23, 1900.

BY J. W. BALLANTYNE, M.D., F.R.C.P.E., F.R.S. (Edin.),

Lecturer on Antenatal Pathology and Teratology in the University of Edinburgh; Honorary Fellow of the American Association of Obstetricians and Gynæcologists.

Gentlemen,—In a lecture delivered here in March, 1899, I attempted to indicate the general scope and possibilities of antenatal therapeutics. Since then some of you who heard that lecture and others who read it in its published form [1] have honored me with inquiries as to individual problems in antenatal therapeutics and with suggestions as to the extension of our therapeutic resources in dealing with morbid states during fœtal life. A consideration of these suggestions and inquiries has led me to believe that many medical men hold erroneous views as to the necessity of treating the unborn child, and that some are inclined to institute unfair comparisons between this and other departments of therapeutics.

The possibility of influencing morbid states that affect the infant while in utero does not cease with its birth. Many malformations which are produced antenatally can be corrected postnatally, and some congenital diseases can be alleviated, if not cured, by therapeutic measures instituted after birth. The treatment of antenatal morbid conditions is not, therefore, exclusively antenatal; it may be, in part, postnatal, and its effects must of necessity be largely postnatal in their manifestation.

I fancy that most physicians, if they think about it at all, compare the therapeutics of diseases of the fœtus with the treatment of

[1] Brit. Med. Journ., April 15, 1899, p. 889; Arch. of Pediat., 1899, xvi. p. 513.

disorders of the adult, doubtless much to the prejudice of the former. They contrast the many medicines which can be administered to the more or less willing adult, with very definite effect, with the very few drugs which can be given, with almost unknown results, to the fœtus in utero, always presupposing that its passive resistance to being drugged at all can be overcome; that, in other words, the placental barriers can be passed. But in so arguing they are not acting quite fairly. Why should comparison be made between the therapeutics of the fœtus and that of the adult? We do not contrast the therapeutics of the new-born infant with that of the adult, but with that of the infant and the child. Let us, therefore, contrast antenatal therapeutics with neonatal therapeutics. I think I am right in supposing that few medical men commonly administer more than two or three drugs to the new-born infant; that few feel altogether at home in its management, medicinal or otherwise; and that few can boast of brilliant results and assured triumphs in the domain of neonatal therapeutics. In comparing neonatal with antenatal therapeutics we compare similar things; the one is divided from the other, it is true, by that epoch-making occurrence, birth, yet they are in many respects similar. A little reflection will make it clear that, after all, antenatal therapeutics in its scope and utility is little if at all behind neonatal therapeutics. In the hands of many medical men both antenatal and neonatal therapeutics may be rated at *nil,* or possibly at *x,* with results which are not, perhaps, so disastrous to the fœtus and neonatus as might be expected.

Further, few drugs are needed by, or commonly administered to, the new-born child, and even these are of doubtful utility: castor oil to do what the colostrum does equally well or better, and dill water to undo the evil effects of unnecessarily filling the infantile stomach with sugar and water. The first draught of the mother's milk thoroughly clears the bowel of meconium. The infant does not come into the world in a state of starvation; its tissues are not calling out for pabulum; it is doubtful if, after birth, the infant ever again receives so full, complete, and well-adapted a meal as is provided for it while still in utero; it requires nothing more for eight hours after birth, by which time the mother's breast is ready to supply its wants. Others add hydrargyrum cum cretâ to the drugs given to the new-born, presumably on the principle that mercury for infants is bound to do good, and cannot, at any rate, do harm; but

is it not somewhat of a reflection upon every one concerned that a child should be started upon his postnatal career with the specific for syphilis, always supposing that it is free from this disease? The most rational plan of giving medicines to the new-born infant is through the mother's milk or by inunction through the skin, which at this time of life absorbs freely; but not many new-born infants require drugs at all. Now, the same general principles apply to antenatal therapeutics. The unborn, like the new-born, infant requires drugs seldom, and he can best receive them through the mother,—*i.e.*, through the placenta, which, after all, is in its fœtal part an extension of the fœtal skin, or ectoderm. In this way arsenic and mercury, and doubtless many other remedies, may be administered; but there is yet much ignorance as to the conditions of the placenta which favor or obstruct the passage of these drugs to and from the fœtus. It may, however, also be said that there is quite as much ignorance in regard to the passage of drugs through the mother's milk to the infant.

In another respect neonatal and antenatal therapeutics may be compared. When an infant is born into the world in a weak, puny, or delicate state, or when it develops weakness or illness soon after birth, the best line of treatment will often consist in attempting to return it to its antenatal surroundings, in re-establishing the *status quo ante partum*. I do not, of course, mean to put the child back in the uterus, but to make its environment as like that in intrauterine life as possible. In its most complete development this attempt finds expression and visible embodiment in the *couveuse*, or incubator, which has of late years become so important an addition to the therapeutic armamentarium of the maternity hospital. The treatment of the new-born is therefore environmental rather than medicinal; so, I maintain, should be the treatment of the unborn.

The fœtus, when healthy, requires no external help; and when ill, no more than is given to the ailing new-born infant by the pediatric or obstetric physician. Indeed, it requires considerably less, for Nature has already carried out a large part of the treatment by providing the best possible *couveuse*, for a fluid medium of constant temperature is better than an atmospheric one, even when artificially warmed and carefully sterilized. When an adult is ill, we order him to keep his bed in a room of even temperature; when

a fœtus is ill, we are glad to know that it is keeping its room. We are embarrassed only if it leave it, for to be prematurely born is a serious matter for a diseased fœtus; and if this should happen, all we can do is to provide another "room" which very imperfectly resembles the uterus,—viz., the incubator. But, it may be asked, Can we in any way aid in keeping the fœtus in utero? I think we can. We can keep down the maternal temperature and prevent a sudden rise, which would loosen the intra-uterine connections; we can assist the mother's excretory organs (skin, kidneys, intestine) to act vigorously and throw off the effete products which pass in large quantity into the maternal from the fœtal economy, for maternal toxæmia will tend to set up uterine action and bring on labor; and, finally, we can do something towards maintaining the structural and functional integrity of the placenta, for if there is one thing in this relation that is fairly well proved it is that chlorate of potassium acts as a placental tonic. How it acts, whether on the placenta directly or indirectly, by keeping the maternal blood in good condition, does not so much matter; in my experience and in that recorded by others it prolongs intra-uterine life, it maintains the placental functions, and so saves the fœtus from the dangers of premature exposure to an extra-uterine environment.

If the death of the fœtus in utero from some chance malady be diagnosed, although there is no longer need for treatment directed towards the fœtus, it may be better for the mother that it remain for a time in the uterus. If, as is common, the membranes are unruptured, the dead fœtus is aseptic, and the puerperium may run its course before the fœtus and membranes are expelled, with diminution in the risk to the mother. When both mother and fœtus are suffering from an infectious disease, it is not the death of the fœtus that endangers the mother's life, but its expulsion from the uterus, for this renders the systemic blood liable to infection through the utero-vaginal canal. In this connection it is well to remember that it is not from the expulsion of the fœtus, but from its death, that the beginning of the puerperium ought to be dated: the involutionary changes in the maternal organism then commence, and are in many cases almost finished before the uterine contents are actually expelled, as I believe they commonly are, about the time of the occurrence of the first menstrual date after the death of the fœtus. Of course, these changes cannot all be accomplished with the fœtus

still in utero, but it is remarkable how nearly the process of involution can be completed.

If these facts be borne in mind, a more correct idea of antenatal therapeutics will be gained. Further, it must not, on this account, be thought that the value of antenatal therapeutics is thereby reduced; its possibilities are simply diminished to a point but little below those of the treatment of the new-born infant. This may be an advantage rather than a disadvantage to the fœtus.

Let us now apply these considerations to the study of the management of some individual cases of antenatal morbid conditions. Suppose a mother has had a typically bad history as regards the antenatal health of the children she has borne; suppose that she has had abortions, premature labors, twins, dead-born or congenitally weak infants, diseased children, and even monstrous fœtuses. Enough is now known of antenatal pathology to make it fairly certain that such a series of repeated antenatal pathological developments points to some such cause as syphilis, alcoholism, infectious or septic trouble, lead poisoning, or other toxic state. If there is reason to suspect syphilis in either parent, treat the mother with mercury and iodide of potassium both before and during her pregnancies, for even if the mother show no sign of the disease, the fœtus in utero may be infected through the father, and may be beneficially acted on by mercury administered through the maternal organism. If the cause be maternal alcoholism, we have no specific for that, such as mercury is for syphilis; but we can remove the specific cause itself,—viz., the alcohol; and recent statistics have shown that apparently the best thing that can happen to the unborn infant of a female alcoholic is for its mother to be consigned to prison for a period of ten months, commencing with the first or second month of its intra-uterine life! The compulsory cutting off of the alcohol may have so beneficent an effect that for the first time in her history the woman may bring an infant to the full term of utero-gestation and send him forth to the world a prison baby, but a living baby.[1]

Again, if the cause appear to be anæmia or some toxic state of the mother (such as that resulting from lead-poisoning), endeavor to improve her health during the pregnancy, and try to remove the

[1] Journal of Mental Science, July, 1899.

cause before she is again confined. A combination of chlorate of potassium and iron may be found useful in such cases. If there be some gynæcological condition, such as endometritis, perimetritis, or parametritis, then the morbid pelvic conditions thus produced ought to be dealt with before another pregnancy supervenes. Finally, if no definite cause can be ascertained, curetting may be advised, in the hope that it may be followed by the development of a new uterine mucosa better able to promote the growth of a healthy placenta and normal membranes; this, of course, is to be carried out between pregnancies. It is possible that the thyroid, which has so marked an influence on the growth of certain tissues, may also have a beneficial effect upon the development of the embryo as a whole, and so be indicated when birth has been given to one or more malformed fœtuses. The administration of thyroid extract or any other drug to a monstripara, with the idea of favorably influencing the development of the embryo as well as the health of the fœtus, does not as yet rest on any sure foundation of clinical experience. It is noteworthy, nevertheless, that both Phillips and Nourse [1] testify to the value of phosphorus in preventing malformations; and the former gives calcium and sodium hypophosphites to the mother during the first six months of pregnancy. It must be borne in mind, however, that the determination of most malformations probably antedates the third month of gestation, and therefore occurs before the time when the drugs referred to can generally be given.

In cases of antenatal morbid states in which there is no history of their previous occurrence there will be no indication for instituting preventive treatment. Nevertheless, a rational treatment of the fœtus does not on this account cease to be possible. If, for instance, a pregnant woman be brought within the range of infection with smallpox, she ought to be vaccinated,—for her own sake if she have not been recently vaccinated, for her fœtus's sake if she have,—for according to some authorities (*e.g.,* Lop [2]), seventy-nine per cent. of unborn infants will be thus protected against variola, as is shown by the fact that that proportion is born immune against vaccinia. If, however, the fœtus be not protected in this way, and there is evidence of its having variola in utero (as indicated by dis-

[1] Brit. Med. Journ., April 29, 1899.

[2] Thèse de Paris, 1893.

ordered fœtal movements, maternal albuminuria, increased rapidity of fœtal heart, etc.), then the treatment of the case should resolve itself into an attempt to prevent the occurrence of premature labor. It is far more likely that the fœtus suffering from smallpox will pass safely through the various stages of that malady in utero than outside the womb, especially since in the latter case the additional risks incident to prematurity will have to be faced. What is known of fœtal variola goes to show that the conditions present are favorable for the recovery of the unborn infant; it is surrounded by a fluid medium of nearly constant temperature, the absence of air is probably a protection against secondary infections, and the exclusion of light doubtless prevents pitting. If the mother be also suffering from variola, there will be all the more reason for saving her from the risks of premature delivery, and therefore the same line of treatment should be pursued. If the smallpox fœtus be unfortunately born prematurely, it should be placed in the incubator and given a chance, albeit a small one, of surviving the disease and the perils of prematurity. In a sentence, what is to be dreaded for the fœtus ill with variola, measles, scarlet fever, typhoid fever, or endocarditis, is being expelled from an environment in every way well adapted to treatment and cure, into one which is, even for a healthy fœtus, full of danger. It is difficult for the healthy infant when prematurely born to survive its birth; for the unhealthy premature infant it is almost impossible. In utero, on the other hand, the wonderful *vis medicatrix naturæ* is afforded every opportunity of efficiently coming into play, and it may bring about recovery under conditions that are apparently almost hopeless. I believe that just as the infant after birth shows this marvellous recuperative power more evidently than the adult, so the fœtus in utero exhibits it even in a more marked fashion than the new-born child. But when premature birth takes place, Nature's therapeutic power is unable to preserve life and restore health; and, as the medical student put it with unconscious humor, and yet with no small degree of truth, " the cause of death is birth."

TREATMENT OF HÆMATEMESIS BY LAVAGE; DIAGNOSTIC USE OF THE STOMACH-TUBE.

BY A. L. BENEDICT, A.M., M.D.,

Professor of Physiology and Digestive Diseases, Dental Department of the University of Buffalo; Special Consultant in Medicine, Buffalo Hospital of the Sisters of Charity.

GENTLEMEN,—This morning I purpose to demonstrate some of the uses of the stomach-tube, an instrument that has been misused and overrated, but which is often of inestimable value. It has been implied, if not actually claimed, that the use of the stomach-tube was purely scientific. It should, however, never be used merely as a matter of routine, but always with a definite conception of what it is to accomplish in the case in question. The indications for its employment may be summed up under two heads,—the extraction of gastric contents for the purpose of examination, and the direct treatment of gastric conditions, either by the removal of offending material or by the introduction of some medicament.

A somewhat rare use of the tube is illustrated in the first case I present to you.

McP., a brakeman, aged forty-three years, was first seen last September. He gave a history of frequently recurring hypochondriac pain and of black vomit, dating back to June. Physical examination showed a dilated stomach, reaching to the umbilicus, with doubtful pyloric tumor and doubtful nodulation along the greater curvature. The patient was lean but not emaciated; sallow but not distinctly cachectic. Indeed, I do not recognize a cachexia pathognomonic of cancer that differs in kind from ordinary sallowness and pallor.

The history of repeated vomiting of black matter was considered reliable evidence of hæmatemesis which, disregarding certain rare possibilities, was doubtless the expression of one of three comparatively common conditions. The usual causes of repeated gastric hemorrhage are, first, chronic peptic ulcer due to digestion of part

of the stomach, the lack of resistance to the digestive process depending either on infarction or local vasomotor spasm, more frequently the latter; secondly, catarrh with hemorrhagic erosions due to portal obstruction; thirdly, ulcerating cancer. The hemorrhages had been rather too small for gastric ulcer, and there were no indications either of vascular disease or of angioneurosis.

Hemorrhagic erosions from portal obstruction are almost always due to sclerosis of the liver, which is usually also contracted. In the present instance, there is a history of alcoholism, which favors the diagnosis of hepatic sclerosis,—though this disease is by no means uncommon in total abstainers,—but the liver area was not markedly diminished. By auscultatory percussion, it was found to extend, and still extends, from the fourth intercostal space to within a very short distance of the free margin of the ribs, the lower limit being verified by palpation, allowance being made for the thickness of the abdominal wall. The hepatic area should extend from the fourth rib, just to the costal arch, but in long-chested persons a liver of normal size may be accommodated within these limits.

The hemorrhage and the symptoms are equally indicative of malignant ulceration; and if I could have been positive of the presence of a tumor, either at the pylorus or on the greater curvature, or if the microscopic examinations had revealed the presence of cancer nests in the stomach contents, a diagnosis of this condition would have been warranted. I have seen at least one case of pyloric tumor, on account of which abdominal section was performed, which proved to be non-malignant, and other cases of the same kind have been reported, so that the mere presence of a tumor cannot be regarded as pathognomonic of cancer. The finding of cancer nests is, theoretically, pathognomonic, but, practically, in examining epithelial remnants that have undergone degeneration or have been partially digested, it is by no means easy to distinguish between a cancer nest and an exfoliated circle of epithelial cells from the mouth of one of the glands, such as we find in gastric catarrh or, occasionally, where there is no organic disease. Personally, I have never found any single nest of cells upon which, alone, I would base the diagnosis of cancer; though, by examining a number of slides, I have been able to make this diagnosis in several cases.

In this case, the diagnosis was undecided between hemorrhagic erosions and ulcerating cancer, very little bleeding occurring after

the case was taken in hand and no typical cell nests being found. I urged an exploratory operation, but this was refused. Accordingly, the obvious catarrh and bleeding were treated by lavage, the introduction of hydrogen peroxide, and, on several occasions, by the use of menthol spray through the tube, on the same principles as in the treatment of a catarrhal throat. The patient made what he considered a good recovery; all symptoms disappeared and great gain in weight and strength followed.

Yesterday he re-entered the hospital, having had three distinct hemorrhages in as many days, but not over two hundred and fifty cubic centimetres of blood were lost at one time. Yesterday the patient was kept quiet and given no food and very little water. Hypodermic injections of morphine and ergot were ordered to be used pro re nata, but they were not needed. To-day he will be brought before you to illustrate one of the rarest uses of the tube. I have been quoted, and rightly, as being opposed to the insertion of the tube in gastric ulcer, the word ulcer being taken in its widest sense. Several years ago, however, while writing on this subject, I admitted that the danger was not from direct traumatism by the soft tube, but from reflex gagging and rise of blood-pressure, and stated, on purely theoretical grounds, that in a patient already accustomed to the tube, so that gagging need not be feared, it might be employed to cleanse and medicate the stomach within a short time after the cessation of a comparatively large hemorrhage. The present case, though probably not one of gastric ulcer in the usual sense, illustrates the exception to the rule of non-interference, and I intend to pass the tube, and introduce small quantities of water at a temperature of 115° F., in order to free the stomach of blood-clots, prevent septic processes which in themselves conduce to further hemorrhage, and take advantage of the well-known styptic and tonic effects of hot water. The patient has taken the tube without gagging or straining, and, as the water runs out, you see that no fresh bleeding has occurred, but that considerable quantities of small, old clots are flowing out. These should be saved for examination. Now the wash-water comes away clear, and a little hydrogen peroxide is introduced as an antiseptic and styptic. The tube is withdrawn and the patient will be sent back to bed with his stomach clean and empty, and in that condition it will be kept, except that fifty centigrammes of bismuth salicylate will be given four times daily as a

powder dressing, not more than five hundred cubic centimetres of water being allowed in twenty-four hours to wash this down and to rinse the mouth. The patient will, of course, receive water and nourishment by the bowel.

You will doubtless ask about the diagnosis. I am still unable to decide between the two conditions already considered, as nothing has developed that could warrant a positive opinion. The duration of the case is rather against cancer, but the exclusion is not positive. I no longer urge operation, because no tumor has developed; and if cancer be present, the process must have already spread insidiously beyond the reach of the knife.

(Note.—The stomach contents were, unfortunately, thrown away. There were no further bleeding and no subsequent local treatment. The patient became so unruly that he had to be nourished by the stomach, and was soon discharged. He reported himself well again about April 1st. The remission makes it probable that the case was one of hemorrhagic erosions occurring in gastric catarrh and due to portal obstruction.)

The next case is one in which, without any positive grounds, there has been some suspicion of cancer. At the same time, especially in a person in middle life, we can never be quite certain that an apparently innocent dyspepsia is not a symptom of beginning malignant disease. For instance, early in December I saw, in consultation, a man about fifty years of age, with a history of chronic alcoholism, who was suffering from chronic gastritis and a contracted liver. The patient and his family, as in the present case, raised the question of cancer. I told them that while there was no indication whatever of cancer, I could not guarantee that cancer was not beginning. Last week I received an invitation to attend the necropsy. The patient had vomited blood frequently during the few weeks immediately preceding his death. Several microscopic examinations had been made, but no cancer nests were found. Physical examination had revealed nothing in any way suggestive of tumor except a vague feeling of resistance in the epigastrium, and before the section the diagnosis was still doubtful. At the post-mortem there were found, in addition to the hepatic sclerosis and gastric catarrh which had been diagnosed, a small ulcerating cancer of the pyloric antrum, extending to the duodenum and pancreas, and multiple secondary metastatic foci in the liver. None of the latter

projected below the margin of the ribs and the former could certainly not have been felt *antemortem.*

Before passing the tube, we make sure that the heart and arteries will stand the reflex rise of blood-pressure, that no false teeth remain in the mouth to fall into the œsophagus or larynx, that the throat is capacious enough to allow the passage of the tube, and that there is no local pharyngeal condition to prevent. I have seen only one case in which the pharynx was so small as to interfere with respiration after the tube was in place, and even then the trouble was due to spasm rather than to actual lack of air-space. Of course, I have occasionally seen syphilitic throats which would not allow the passage of the tube; but none of these presented indications for interference with the stomach. We must also consider the presence of pregnancy, hernia, hemorrhoids, hysteria, etc.

To introduce the tube, the patient is instructed to hold the head in the ordinary position, not throwing it back as might seem better, to swallow hard whenever there is a tendency to gagging, and to make free use of Nature's anæsthetic and antispasmodic, panting. With these precautions, the tube passes without much difficulty, though it is the patient's first experience. The stomach contents are removed, partly by expression, partly by suction. Most gastro-enterologists use some form of aspirator bottle or a Politzer bulb. Personally, I have devised this roller tube-stripper, which acts on the principle of the Allen surgical pump, the rebound of the walls of the rubber tube causing a vacuum. It is obvious that a powerful aspirator of any kind should never be used, and even these gentle measures sometimes draw a little blood. Some difficulty is experienced in getting the stomach contents. I cannot explain this; but it frequently happens that all rules of siphonage are apparently broken, especially at the first *séance.* Sometimes, the trouble is due to the lodgement of particles of unmasticated food; but often this explanation does not hold, and I have sometimes thought that the tube was compressed by a spasm of the cardiac sphincter, though this would hardly seem probable.

An hour and a quarter ago the patient took a test-breakfast, consisting of fifty grammes of dry bread, five grammes of butter, and two hundred and fifty cubic centimetres of water. The quantitative tests for acidity are best applied to the unfiltered chyme, as the particles of undissolved food hold a good deal of acid; other-

wise the filtered contents are used. Both the quantitative tests and the process of filtration are somewhat tedious, so I have brought with me the stomach contents of a patient, who consulted me yesterday, in order to demonstrate the qualitative tests. If a drop or two of filtered chyme is added to an inch of milk in a test-tube, and kept at the body temperature for five or ten minutes, the rennet ferment causes coagulation. This is a rough test as to the digestive power of the stomach, as, commonly, though not invariably, rennet and pepsin rise and fall together. The absence of ferments in the stomach is not positive proof of any one disease; but, if constant, it points either to disappearance of the gastric glands,—anadenia,—or to severe functional derangement, such as is found in advanced cancer, in Addison's disease, in severe catarrh without actual anadenia, and, in general, in all states of extreme depression. You notice that I have avoided the expression "achylia gastrica." Anadenia,—that is, atrophy of glands of the stomach,—necessarily entails achylia gastrica,—that is, failure of gastric secretion. I have not the slightest objection to the latter term, except that it is sometimes understood to mean some special form of gastric disease. I prefer, however, to say that this or that function of the stomach is normal, defective, or excessive, and avoid the use of such technical terms as achylia, hyperchlorhydria, etc., to express the same meaning. The compact Greek terms are very convenient; but they have led to a quite general misconception that they represent pathological entities instead of conditions.

(Note.—The patient from whom the gastric contents had been taken proved to have very nearly normal secretory and chemical conditions of the stomach.)

ON THE TREATMENT OF DIPHTHERIA.

BY HUBERT E. J. BISS, M.A., M.B. (Cantab.), D.P.H.,

Assistant Medical Officer to the Grove Fever Hospital (Metropolitan Asylums Board).

Diphtheria is pathologically a true toxæmia. In toxæmic diseases two morbid conditions are present, the local elaboration of toxines, and their distribution throughout the system by the circulating fluids. In combating a disease of this character one of two methods may be chosen,—the local source of the toxines may be attacked, or an attempt may be made to neutralize the toxines that have already found their way into the circulation. Both of these methods may, however, be combined.

The specific treatment of diphtheria from the time of Trousseau till the early years of the present decade has consisted in employing the former tactics. Every means was called into requisition that seemed likely to accomplish the destruction or to prevent the reformation of the local membranous exudation. Escharotics, the cautery, antiseptics, were successively employed, but with little success. The ravages of this awful disease continued despite the most vigorous of such measures.

It is not our intention to point out the revolution in practice that accompanied the recognition of the true pathology of this disease, although it forms one of the most brilliant pages in the history of rational medicine. Within their own generation a body of investigators have seen the fruition of their labors in the saving of thousands of lives by methods as exactly scientific and as little empirical as possible.

Now, in what way was this accomplished? It was by supplying to the tissues of the diphtheria patient a substance capable of checking the reception of toxines at a definite point. It can do more than this: it can so reinforce the local cells at the point of toxine production that a limit is imposed to their elaboration; still more, it can confer temporary immunity from the attack upon a

non-invaded subject. Such are the functions of the diphtheria-antitoxic serum.

In the blood of a horse that has been repeatedly inoculated with diphtheria-toxine an antitoxin is elaborated; the serum is separated from the blood of a horse treated in this way, and is standardized on guinea-pigs to ascertain the number of immunizing units of antitoxin present; it is then ready for use as a protective and curative agent.

This serum is specific in regard to diphtheria; that is to say, it is capable of directly neutralizing diphtheria-toxine. Exactly how the antitoxin antagonizes the toxine is still a question on which opinions differ. The disputants may be regarded as ranged in two camps, the camp of Behring and the camp of Büchner. Behring regards antitoxin as the product of the immunized animal's tissue-cells; he maintains that it is a direct chemical neutralizer of toxine; comparing this action to that of an alkali on an acid. Büchner holds that antitoxin is a bacterial product very similar to toxine, and capable of combining with animal tissue-cells to the exclusion of toxine. Whichever be the correct view, two points are clear: first, that antitoxin is a substance that has the power of rendering animal tissue-cells immune for a certain time (probably only a few weeks) to toxine; second, that antitoxin *may* also have the power of directly neutralizing the free toxines with which it comes in contact.

From this it follows that, since diphtheria is a toxic disease, and the function of diphtheria-antitoxin is so to reinforce the tissue-cells as to make them invulnerable to diphtheria-toxine invasion, it will be impossible for antitoxin to undo any harm that toxine may have already done to any cell or cells. The recognition of this proposition is of the utmost importance in the therapeutics of diphtheria-antitoxin.

The subtle toxines of the Klebs-Loeffler bacillus combine with certain cells, especially those of the nerves, the heart, and the gastric mucous membrane, and cause their rapid degeneration. Antitoxin cannot undo this mischief: what it can and does do is to prevent any further combinations of toxines with tissue-cells. The obvious conclusion from these facts furnishes the most important therapeutical indication: *antitoxin should be given at the earliest possible moment.*

On what principles should dosage be regulated?

Antitoxin is, in itself, harmless; no recognizable harm can possibly be traced to it. The serum in which it is held contains certain other bodies,—ferments probably,—which do produce certain unpleasant effects. The chief of these are pain in the muscles and joints, and erythematous and urticarial rashes. Though unpleasant, irritating, and painful, these are never of serious moment. Such manifestations do not necessarily follow inoculation; curiously, too, their intensity is not proportionate to the dose given.

The deduction from these considerations carries with it the next indication: *a sufficiency of antitoxin should always be given; the error should always lie on the side of over-dosage rather than under-dosage.*

The next question that presents itself is this: Should antitoxin be given in a series of moderate doses, or should all that is considered necessary be supplied at one injection? Bearing in mind that the object is to immunize the uninjured cells at the earliest possible moment, and that, practically speaking, too much antitoxic serum cannot be given, there can be but one answer.

Consider the case; determine the dose and fearlessly give a fully sufficient quantity of antitoxin at the earliest opportunity. The patient's tissue-cells will then be rendered immune to any further toxine invasion as soon as the antitoxin has taken effect. To give any further dose is to cause needless inconvenience and pain.

Antitoxin probably takes some hours to act; but the disease is cut short at the point it has reached when the antitoxin takes effect. Regard must be paid to this contingency: the patient may still die from the effects of the poison absorbed before the antitoxin was given. Such harm cannot be undone; further harm only can be averted.

To consider dosage more exactly. Bottles of serum are supplied containing certain quantities of immunizing units. The strongest serum,—that is, the serum containing the largest number of units in a given bulk,—is the most convenient for use and the least distressing to the patient. Measuring the dosage by these units, there are three chief considerations that should influence the decision as to the number to be employed:

1. *The Day of the Disease.*—For an ordinary mild case on the first day of disease, two thousand units will probably suffice. At

any rate, that should be the minimum. The later in the disease the larger the number of units that should be given; the value of antitoxin is inversely proportionate to the length of time the disease has existed. It is very doubtful if antitoxin has any value after the fifth day; by that time the disease will have advanced to such a point that it is extremely unlikely that any quantity of antitoxin can appreciably affect it. Still, if there be any local process to indicate active toxine elaboration, the patient should have the benefit that may accrue from inoculation, however late the disease be seen.

Cæteris paribus, the longer the disease has existed the larger should be the dose of antitoxin. If there be any hesitation between giving, say, eight thousand or twelve thousand units, a good rule is to give the larger dose. It is making assurance doubly sure.

Is there any limit to the amount that should be administered? Enormous doses have been given and good results obtained. Still, there would seem to be a maximum beyond which it is unnecessary to go. Twenty thousand units is about that maximum; in other words, what twenty thousand cannot accomplish no number of units seems capable of effecting.

2. *The Age of the Patient.*—Diphtheria is an infinitely more dangerous disease to young children than it is to adults. The younger the child the more necessary will it be to immunize effectively its unaltered tissues to toxine invasion. The relatively larger doses must be for the young.

Some practitioners withhold antitoxin from adult patients; this does not seem a wise course. Diphtheria causes death and its other sequelæ in adults; there is no means of telling how susceptible any particular individual may be; antitoxic serum can cause only temporary inconvenience at most. There would seem, then, to be no legitimate reason for withholding its benefits from adults, even though they incur its possible annoyances.

3. *The Severity of the Disease.*—This is judged of by the extent of the local membrane, the rapidity with which it has formed, and by the constitutional effects of the toxæmia. Individuals that have a severe attack of diphtheria possess low resisting powers to the disease. It is, therefore, necessary to reinforce their tissue-cells with a larger number of immunizing units than is required by a relatively insusceptible patient.

It is impossible here to deal with the indications to be met in

every phase of diphtheria in every kind of individual. An adult with a small patch of glistening, pearly membrane on one tonsil, and but little systemic disturbance, on the first or second day of the disease may be treated with from two thousand to four thousand units; while a child of two or three years, with fauces, palate, and nasal mucous membrane clothed in shaggy, greenish membrane, much submaxillary lymphadenitis and periadenitis, and severe constitutional symptoms, who has been ill four or five days, should receive twenty thousand units. Between these extremes there is every conceivable gradation.

Specific treatment having been instituted, there are certain general considerations to which attention should be turned.

Isolation is essential. That, however, is not the only preventive measure that should be employed. If there be other children in the house, they may be already infected, or may shortly fall a prey to the same influences to which the patient has succumbed. The situation should always be explained to parents, and the offer made to immunize them by injection of antitoxin. A small dose, say, five hundred units, will be sufficient for this purpose. The immunity given is short, probably about three weeks, but it will suffice to tide them over a period of special danger.

The sick-room should be of good size, with ample light and good ventilation. The danger of cardiac failure must never be lost sight of; a patient suffering from even the mildest form of diphtheria should be put to bed and kept recumbent for three weeks. During the pyrexial period the diet should consist of milk, and plenty of it; it should be given in small quantities at short intervals; but when the temperature has fallen and the tongue has become clean, it is well to pass on quickly to a generous dietary. In the adynamic condition left by diphtheria,—a condition that may be followed by dangerous sequelæ,—the sooner the patient is helped to recover lost ground the better. Boiled fish, milk-pudding, bread and butter, may be soon allowed; they may have to be subsequently withdrawn.

Is local treatment,—treatment directed to the alleviation of the faucial condition,—desirable? Though at first sight this may seem of great moment, it is attended by certain disadvantages which cannot be overlooked. Convinced that antitoxin will effectually cut short the disease at the point it has reached, the local condition may be regarded merely as a source of some discomfort. A balance must

then be struck between the relative discomfort of cleansing the throat and that attending the normal process of separation of the membrane. Having enjoined rest, absolute rest, as of the chiefest moment, it is apparent that a struggle, renewed every three or four hours, with a rebellious child, for the purpose of syringing, swabbing, or spraying the throat, is a very dangerous procedure. An intelligent child of eight or nine years may,—an adult doubtless will,—appreciate the benefit of removing the slimy mucus and particles of membrane that cause so much discomfort. In such cases syringing or gargling with a warm solution of weak chlorinated soda gives pleasant relief. Children under seven generally resist local applications with considerable vehemence. The danger of throwing an extra strain on a specially taxed heart; the fact that the membrane-clad throat is not a very sore throat; the knowledge that young children take their milk very well with most extensive faucial manifestations,—all these considerations far outweigh any arguments for persisting in the mere toilet of the throat. Nasal syringing, except when a chronic rhinorrhœa is present during convalescence, should be discarded for the same reasons.

Pyrexia practically never calls for special measures for its reduction.

Severe toxæmia with flagging heart will need to be combated with strychnine and brandy; the latter agent has, however, but a very limited range of usefulness in diphtheria.

When the membrane is confined to the larynx, or has spread to that organ from adjacent structures, early recourse must be had to antitoxin. When respiration is embarrassed, the dyspnœa may be relieved by placing the patient in a warm, moist atmosphere and fomenting frequently over the region of the larynx.

The steam-tent as usually applied is useless and dangerous. An attempt to breathe the steam-saturated atmosphere for a few moments will soon convince any unprejudiced observer of the difficulty of respiration under such circumstances. Placing the cot in a well-ventilated spot, with the steam-jet from a bronchitis-kettle playing *above* the child's head, will render the air a little warmer and a little moister. Warm, fresh, moist air is not so irritating to the respiratory passages as quite cold air. That is all that is needed. If by these measures operative interference can be warded off for twenty-four hours, the necessity for it will probably have ceased

to exist. By that time the antitoxin will have done its work, and membrane will have ceased to form, and will not improbably have begun to separate. Urgent dyspnœa, not controlled by these means, will call for tracheotomy or intubation.

Complications.—Foremost among these is that dreaded attendant of diphtheritic toxæmia, heart failure. This complication, though not regarded as a usual one, is of sufficient importance and frequency to warrant its being dealt with by itself. The heart may give out when the toxæmia is at its height, but that is not the heart failure meant. The condition referred to is that which arises sometimes within a day or two after the subsidence of the acute symptoms; sometimes a week, fortnight, or even longer after the pyrexial period. In the lull after the storm of the disease the child may seem well, though weak. Great care is requisite during this period. One day he is noticed to be quiet, not so much interested in his surroundings, less inclined to play. Soon he vomits, vomits again, is pale, languid, uneasily quiet, quietly restless. Examination of the heart will show that it is perhaps dilated, unusually rapid or abnormally slow, frequently irregular or intermittent. In a few hours, a day or two, the patient is dead. The pathology of this condition is variously described. Certain it is that microscopic examination will seldom fail to reveal irregularly disposed patches of fatty degeneration in the heart muscle and in the mucous tissues of the stomach.

Much may often be done by judicious treatment for a child in this state. A strict guard must be kept to maintain a recumbent position. Bearing in mind the pathological condition of the stomach, no attempt should be made to give food by the mouth. Rectal alimentation is wonderfully well borne if skilfully applied: children may be kept alive for weeks by it alone. It is well worth careful supervision of detail. If the albuminous constituents of the foods administered are to be absorbed, they must be thoroughly predigested; unless the albumens are transformed into peptones they cannot be utilized. So long as milk and the excellent peptonizing powders on the market are available, there is no need of employing manufactured nutrient suppositories, which may or may not be all that is claimed for them. The peptonization must, however, be thorough and complete: an hour or an hour and a half is not too long to allow for this purpose. Testing the milk with a little acetic

acid or lemon-juice is well worth the trouble; it may save one the chagrin of finding, at a post-mortem, the rectum and sigmoid flexure filled with desiccated curd. For a child three or four years old six ounces of peptonized milk should be given every four hours. The method of its administration, too, must not be neglected. Nothing is more mortifying than to have these enemata, on which the child's sustenance depends, returned by the rectum. An hour before they are to be given the rectum should be washed out by a simple enema. Next, take a soft rubber catheter whose upper end is connected with a receiver; the barrel of a glass-syringe, from which the piston has been removed, answers admirably. Anoint the catheter with some lubricant. With the patient on his left side, hips and knees flexed, gently insert the free end into the anus, and let it glide slowly up the bowel for eight or nine inches. Into the empty syringe-cylinder pour, little by little, the peptonized milk (warmed to body-temperature), and allow it to gravitate into the rectum. When the process is over gently withdraw the catheter, and press a warm, folded napkin against the anus. The child must be kept very quiet during the administration of the enemata. By such means it may be safely predicted that a degree of success will be attained in the retention of the enemata such as cannot be obtained with syringes, whose nozzles irritate the sensitive portion of the rectum, and whose contents, when squirted in suddenly, balloon out its lower segment.

The exceedingly low blood-pressure and the ill-filled arteries at this stage of the disease call for measures for their relief. As nothing should be given by the mouth except occasional sips of warm water to relieve pressing thirst, the rectum must again receive the proposed remedies. A very good plan is to give water enemata, at body temperature, of similar size, at like intervals, and administered in the same manner as the nutrient enemata. Such water enemata form convenient vehicles for drugs. Thus the patient will have every two hours an enema of peptonized milk, alternating with water containing drugs.

Drugs innumerable may be used; but there is one that is so pre-eminently useful that it should be employed to the exclusion of all others. This is strychnine. Introducing it by rectum, too, is far more satisfactory with children than giving it hypodermically. Brandy may be given in the same way if the rectum be not intoler-

ant of it; it is, however, very difficult to assign any positive value to the action of this remedy.

The paralyses that follow the action of diphtheria-toxine on the central nervous system and the peripheral nerves are now to be considered. It is comforting to know that the regeneration of the nervous tissue is only a matter of time,—from the clinical point of view. If the paralyzed structure do not directly interfere with a vital process, rest in bed and strychnine are the only measures necessary. In the case of prolonged paralyses of limb muscles, faradism twice a day, for a quarter of an hour, will help to maintain their functional activity.

The two forms of paralysis that call for special treatment are the common palatal paralysis and the rarer diaphragmatic paralysis. In the former case it is the discomfort caused by the return of fluids through the nose, and the difficulty of nourishing the patient, that demand particular measures. A slight paresis of the palatal muscles, not attended with much regurgitation of fluids through the nose, may often be controlled by the simple device of thickening the child's milk, to the consistence of gruel, with corn-flour or arrow-root. More severe cases, and those in which there is paresis of other pharyngeal muscles, will require all fluid nourishment to be administered by a nasal tube. The device recommended for rectal feeding may be used for this purpose also. Such dexterity may be attained in manipulating it that a child may often be fed during sleep. Difficulties are sometimes met with due to rhinitis in a small nose, or to adenoid vegetations in the naso-pharynx. If either of these absolutely preclude the use of the nasal tube, a stouter tube must be passed directly through the mouth into the pharynx; a rather more difficult procedure.

Paralysis of the diaphragm is a terrible complication, and, happily, a rare one. The immediate result of this occurrence is inability to get rid of the bronchial secretions: this is followed by œdema of the lungs. The child's actual danger is that of being suffocated by its own secretions. The expulsion of fluid may be facilitated by raising the foot of the cot; the quantity of the secretion may be diminished by the exhibition of atropine; the action of the respiratory centres may be stimulated by strychnine. These measures, together with rectal alimentation, are worth trying. They are sometimes attended with success.

Otitis media, lobar and lobular pneumonia, nephritis, albuminuria, and submaxillary lymphadenitis may also be encountered. They should be treated on general principles.

Occasionally a relapse occurs; a few weeks after the subsidence of the symptoms membrane may again form in the fauces. It has been observed that if antitoxic serum be injected under these circumstances, after having already been given for the primary attack, certain unpleasant symptoms may follow. Pyrexia, an erythematous or an urticarial rash, faintness, and sometimes, but rarely, alarming collapse may set in within a few hours. Though no such cases have been known to terminate fatally, the possibility of the occurrence of these symptoms should be borne in mind.

THE TREATMENT OF CATARRHAL ICTERUS.

CLINICAL LECTURE DELIVERED AT THE NEW YORK POLYCLINIC HOSPITAL AND MEDICAL SCHOOL FOR GRADUATES.

BY JAMES J. WALSH, M.D.,

Instructor in Diseases of the Chest and Internal Medicine at the Polyclinic, etc.

GENTLEMEN,—After the clinic last Saturday one of your number came to me for advice as to some symptoms from which he himself was suffering. As his case proved to be one of much practical importance, I propose to devote some time to it to-day. He stated that for some eight or ten days he had been feeling out of sorts, —" not himself," was his expression. He had lost appetite almost completely; for meat in particular he had not the slightest desire; and the mere thought of fat caused a loathing for food. He had been constipated for some days, and felt dull and drowsy. He could scarcely keep awake during lectures,—but that is not a symptom of very grave import in most cases,—and he found application to study extremely difficult. Finally, for the last two or three days he noticed that he was becoming yellow. While the jaundice was by no means marked, and in a complexion naturally dark like his might easily have passed unnoticed, a glance at his conjunctivæ made its presence unmistakable. We have here, then, a case of icterus.

Fifty years ago jaundice was regarded as an independent disease, and was set down in the text-books under the Latin name of *morbus regius*. Now we recognize at least a dozen different varieties of jaundice, or icterus, as it has come to be called, none of which is regarded as primarily an independent affection, but merely as the symptomatic expression of some pathological process that either hinders the flow of bile or in some other way causes the resorption of its constituents into the blood. Among the more important varieties of icterus may be mentioned the catarrhal form, which is due to a simple inflammatory condition of the duodenum involving the papilla at the point of entrance of the common bile-duct into the intestines, with swelling of the mucous membrane of

the duct and consequent diminution of its lumen, and damming of bile in the liver; icterus from closure of the bile-duct by stone in the biliary tract, and icterus from obstruction by tumors, usually cancerous, involving the liver or the bile-ducts.

Besides these more common forms of icterus there are certain other types that should be kept in mind, to avoid possible errors of diagnosis. A not unusual form of icterus, icterus menstrualis, occurs at the menstrual period, almost regularly in some patients. It is usually very slight in degree, but it is sufficiently noticeable in delicate blondes to be a source of annoyance. Senator, who has had a good deal of experience with the affection, attributes it to a vicarious hyperæmia of the liver. It can often be lessened or even prevented by the free use of salines just before the menstrual period. Icterus ex emotione,—that is, icterus due to strong emotion,—was described before the Christian era, and to the knowledge of its occurrence is doubtless referable the saying that the angry man has bile on the liver. Hemorrhagic icterus, the jaundice which develops as a result of hemorrhage especially into one of the cavities of the body, with consequent resorption of biliary coloring-matters from the blood, is well known.

Icterus toxicus,—that is, jaundice due to various poisons,—is also an important type of the affection to remember. It used to be said that it occurred after the administration of chloroform, ether, and even chloral hydrate; but very few such cases are reported any more, and it is probable that some other cause, perhaps slight infection after operations in which anæsthetics were employed, provoked the jaundice in these cases. The toxines from certain varieties of mushrooms are known to produce acute icterus, this being a typical symptom of their ingestion. Phosphorus, of course, causes intense jaundice, and death from fatty degeneration of the liver. Arsenic in very small doses in certain susceptible individuals produces distinct icterus. The soluble salts of mercury are said to have the same effect, and chronic lead-poisoning is sometimes attended by a light form of jaundice.

The form of jaundice of greatest importance to the general practitioner, however, is catarrhal icterus, a rather typical example of which we have in the case before us. The history of such cases, while by no means identical in different patients, is sufficiently uniform to make the affection easy of recognition. The attack is nearly

always preceded by symptoms of indigestion, sometimes gastric in character and acute, but more often indefinite, and pointing rather to involvement of the small intestine. These symptoms not infrequently develop after excesses in eating or drinking, or after partaking of food which does not ordinarily agree with the patient. The offending material usually is of a fatty nature.

Exposure to damp cold seems to be an important causative factor in most cases. The disease is known to develop most frequently in the spring and in the fall. In damp years it is so common in Paris during the autumn as to have given rise to such expressions as, "It rains jaundice at Paris this fall." Just how much cold has to do with the causation of this disease is, as in many other affections referred to it, rather difficult to determine. It is probable, however, that deficient sunlight and increased moisture in the spring and fall play a more important *rôle* than low temperature, as they furnish favorable conditions for microbic growth, and the intestinal catarrh which is recognized as the direct cause of jaundice is doubtless of bacterial origin. Catarrhal icterus sometimes occurs epidemically in barracks and the like.

The first symptoms of the disease are not easy of recognition. The patient will often complain merely of general malaise and discomfort, with loss of appetite and symptoms of indigestion; and while he is being treated for this condition will himself call the physician's attention to the development of jaundice. It is well, then, to be on the lookout for the very earliest manifestation of icterus. In most cases this can be first detected in the white of the eye. Before any deposit of bile pigment takes place in the tissues, the blood is distinctly discolored, and this can be seen on the sclera. It must not be forgotten, however, that a certain amount of yellowish discoloration of the sclera is normally present in some individuals. When deposit of bile pigment begins, the discoloration is brought out in the conjunctivæ by contrast with the white sclera beneath. There are, however, some cases in which this is not true, and in these search must be made elsewhere. The next most favorable regions for early detection of icteric discoloration are the nasolabial folds, the angles of the mouth, and the forehead.

The mucous membrane, as well as the skin, shares in the yellowish discoloration. As a rule, it cannot be seen on the mucous surfaces until after it has been plainly visible for some time on the

skin in the regions of predilection. Sometimes, however, the discoloration on the mucous membranes appears early. The icteric shade can then be best seen in the mouth just over the hard palate, where the mucous membrane is thin. If the patient be made to open the mouth widely, the mucous membrane will be rendered tense, especially for a short distance along either side of the median line of the hard palate, the blood will be forced out of the tissues to a greater or less degree, and it will be easy to see any yellowish discoloration that may be present. In general, to make the change of color apparent in the mucous membranes it is necessary to force the blood out of them. This may be done by pressing upon the lips with a glass spatula or a microscope slide.

Besides the jaundice, there are two other symptoms that help to confirm the diagnosis of catarrhal icterus,—a uniform enlargement of the liver and an enlargement of the spleen. The backing up of bile in the liver causes an irritative hyperæmia. This increases the area of hepatic dulness, and gives rise to a subjective sense of pressure and fulness over the region of the liver and tenderness, especially to deep palpation. The enlargement of the spleen is due in part to the interference with the portal circulation in the liver, and in part to hyperæmia of the spleen due to the irritation caused by the presence of bile. A point of the greatest importance in physical diagnosis is the sympathetic relation between the liver and spleen. When the liver is affected the spleen will almost invariably be found enlarged. So true is this, that extreme hesitation is now the rule in diagnosing a cirrhosis of the liver if an enlargement of the spleen be not demonstrable. It is generally conceded that these two organs are very intimately related, not only through the portal circulation, but also by nervous bonds in the sympathetic system.

Two sources of error must be carefully guarded against in making the diagnosis catarrhal jaundice. Icterus in patients somewhat advanced in years, though preceded by digestive symptoms and apparently developing acutely, is very often due to cancer of the liver. Primary cancer of the liver is rare; but secondary cancer in males, who are especially liable to catarrhal jaundice, is very frequent. The occurrence of jaundice, then, at this time of life, makes a careful examination as to the existence of cancer imperative. Cancer of the stomach, cancer of the rectum, cancer of the prostate, cancer of the uterus, should be eliminated before the diag-

nosis of catarrhal jaundice is positively announced. Even then it will often be found that the so-called catarrhal jaundice is really due to latent cancer.

The other source of error is gall-stone. Generally, it is easy to eliminate mistakes of diagnosis due to this cause; but in certain cases it is practically impossible. Gall-stones sometimes exist in the gall-bladder without producing any symptoms indicative of their presence. Occasionally they excite catarrhal conditions or predispose to infection from the intestine which sets up catarrhal inflammation of the bile-ducts, or their presence may excite spasm of the ducts with the same result. Sometimes the passage of a stone through the bile-ducts is attended by no definite symptom. In a number of reported cases gall-stones large enough to cause intestinal obstruction have been removed from the intestine when there had been nothing in the history of the patient to indicate their existence.

It is often necessary to make a diagnosis at once, in order to be able to tell the patient the course that the affection will probably pursue. It must not be forgotten, then, that bile may be detected in the urine when the jaundice is so slight as to be practically unrecognizable. To determine the presence of bile in urine when the jaundice is at its height is very easy. The dark, portery urine whose froth is of a distinctly yellowish tinge is characteristic. In less pronounced cases it can be readily detected by the nitric-acid test. If a certain amount of crude nitric acid,—that is, nitric acid to which some nitrous acid has been added,—be placed at the bottom of a test-tube and the suspected urine poured gently over it, a play of colors,—green, blue, yellow, and red, finally passing into brown,—will be observed at the point of contact if bile be present. It must be remembered that green is the important color in this test; and that if it be absent the test is not positive.

For very small quantities of bile in the urine, the filter-paper test is best. A considerable quantity of urine, say, from six to eight ounces, is allowed to pass through a piece of white filter-paper. This is then spread on a plate and touched with a glass rod that has been dipped in crude nitric acid. A play of colors develops, green forming the outermost ring. This test is very delicate, and yields positive evidence of the presence of bile-coloring matter in the urine, and consequently in the circulation, before there is any other

definite indication of occlusion of the biliary tract or of absorption of bile.

As soon as the diagnosis has been made, careful treatment should be insisted upon. The affection usually lasts several weeks; sometimes it persists for several months; or it may become chronic and last for years. Its duration depends to a large extent upon the treatment of the condition before it has become chronic. Every source of irritation of the intestine should be removed. The diet should be of the blandest and most unirritating character. For the first few days an absolute milk diet should be enjoined.

After four or five days, eggs slightly cooked and beaten up with the milk may be added to the diet. To this a little alcohol may be added with advantage. The irritation of nerve-centres produced by the presence of bile in the circulation causes a slowing of the heart. This retards metabolism throughout the body and entails impaired function. Stimulation is required. After the lapse of a week, if the icterus still continues, as it usually does, starches and sugar may be added to the diet. They are usually well borne, and amply make up for the deficiency of fats.

Usually this diet will suffice to maintain the patient's normal condition and weight until the attack has passed off. Sometimes, however, eggs and milk are not well borne, and the starches and sugars may cause flatulence and meteorism. In that case it will be necessary to allow a more liberal diet. Sometimes the vegetable oils, such as olive oil, will be well borne, even in considerable quantities, when other fats produce nausea or indigestion. When meat is given, the white meat of fowls and the like should be selected; it should be well cooked. Rare steak and roast beef will usually be found to be indigestible.

Notwithstanding care in the diet, symptoms pointing to the absence of bile in the intestines will often be noticed. An important function of bile is that of an antiseptic. The specific indication, then, when no bile escapes into the intestine is to provide a substitute for it with similar antiseptic properties. For this purpose calomel has been recommended for many years. Hanot recommends giving five to ten grains of calomel every day for eight days, and then, after a pause of eight days, giving it once more for the same period, continuing this treatment until the jaundice disappears. Of late years, instead of calomel other intestinal antiseptics

have been proposed. Salicylate of sodium, in doses of ten to fifteen grains three times a day, or salol, may be given in about the same way. Naphthol has also been recommended. Some time ago animal bile was suggested as a substitute for the natural secretion of the liver. It has been used with good results. It has been found, however, that one of the biliary salts produces the same effect as the bile itself. Sodium cholein in doses of from five to twenty grains a day gives very good results, and is much more palatable than the bile.

Certain mechanical methods of treatment have been suggested to shorten the duration of the icterus. Of these, the manœuvre suggested by Gerhardt, and used by him with success in a number of cases, is the most promising. He recommends firm massage over the region of the gall-bladder in order to press some of the gall into the intestine through the duct. This procedure often forces out the plug of mucus which occludes the bile-duct during the inflammatory stage and puts an end to the condition at once. The resistance offered by the obstruction has been felt to give way during the manipulation, the stools are colored by bile a few hours afterwards, the jaundice immediately begins to diminish, and finally it disappears entirely. Although this method of treatment is successful in only a certain proportion of cases, the results obtained warrant its employment in all cases of catarrhal jaundice.

Another form of mechanical treatment is the administration of clysters. These are given with two objects in view,—to increase peristalsis and to empty the colon. If it is desired to increase peristalsis, cold water is given and in rather large quantities,—two to three pints. The effect of increased peristalsis is to produce a gentle massage of the intestine that reduces the chronic inflammation; and as this action is supposed to extend to the duodenum, it is hoped that the inflammatory swelling occluding the bile-duct may also be relieved. The other object for which clysters are given is to remove the contents of the large intestine, in which, owing to the absence of bile, there is likely to be offensive putrefaction. For this purpose clysters of half a pint of lukewarm water are given two or three times a day. The water is allowed to run in slowly, and should be retained as long as possible. Some simple antiseptic like boric acid may be added to it. The gentle increase of peristalsis

caused by this method of treatment is also claimed by certain clinicians to decrease materially the duration of the icteric condition.

Constipation should not be allowed to continue for any length of time. To overcome it the salines, especially the phosphate of sodium, were formerly recommended. The tendency now is to use the vegetable laxatives for this purpose. Of these, rhubarb, licorice, or senna is especially recommended. When starches and sugars are not well borne and fermentation causes pain, often accompanied by constipation, the use of fresh beer yeast has been found very serviceable. It is given in doses of from thirty to sixty cubic centimetres a day in pure culture.

Certain symptoms which usually accompany icterus require treatment. Reference has already been made to the slowing of the heart and of the pulse. The lessened metabolism consequent upon this condition usually causes a lowering of the temperature of from three-tenths to five-tenths of a degree, and the patient complains of feeling drowsy and tired. Much of this uncomfortable feeling can be removed by stimulation. Atropine has been found especially useful in increasing the pulse-rate, and this relieves the subjective symptoms of which mention has been made.

Patients often complain of an extremely bad taste in the mouth. For this, particularly when the tongue is coated, the mineral acids, especially when combined with a bitter tonic, will be found of very great service; the following prescription may be given:

℞ Acid. hydrochlor. dilut.,
Acid. nitrici dilut., āā ʒj;
Tr. nuc. vomicæ, fʒij;
Tr. gent. comp., q.s. ad fʒiij. M.
Sig.—A teaspoonful every three or four hours.

A saturated solution of borate of sodium in peppermint water, or in water scented with a little oil of cloves, may be used as a mouth-wash.

Pruritus is a very common complaint. Lesions of the skin are often produced by scratching. The itching is most annoying on the palms of the hands, the soles of the feet, and between the fingers and toes. When it becomes a serious annoyance large doses of the bromides, thirty to forty grains, morning and evening, may be required to relieve it. External applications often suffice to over-

come it. Lemon-juice has been recommended, or a two per cent. carbolic acid solution; diluted acetic acid or vinegar often answers well. Carbolic ointments are sometimes even more effectual than washes. Eichhorst recommends the following prescription:

℞ Acidi carbolici, ʒss to ʒj;
Lanolini,
Adipis suillæ, āā ʒss.
Sig.—For external use.

Warm baths are very soothing to patients suffering from icterus, and should be used frequently. They excite cutaneous activity and cause the external layers of the epidermis to be thrown off, and so promote more active elimination of deposits of biliary pigment. The addition of a certain amount of bicarbonate of sodium or sea-salt to the bath has been recommended. Whether these substances really do good is doubtful, but patients are willing to take the baths more frequently when such additions are made to them.

A serious symptom in many cases is the sleeplessness that develops during the course of the disease. The theory is that bile in the cells of the cerebrum acts as an irritant and keeps these from getting their normal rest. Certain it is that in many cases the sleeplessness is one of the most distressing features of the affection. The remedy usually recommended for it is chloral. If pruritus be also present, both symptoms may be combated by the use of reasonably large doses of a bromide. The warm baths which alleviate the pruritus are also an excellent remedy for the sleeplessness. They should be taken immediately before retiring, and the patient should be instructed to get into bed without drying his body. This treatment also serves a very useful purpose in aiding the kidneys to eliminate a certain amount of extraneous material which finds its way into the circulation during the existence of the icterus.

The state of the kidneys should not be neglected, especially if the icterus has lasted for some time. The irritative material that the kidneys are constantly called upon to excrete may set up serious inflammatory changes in these organs. The urine should then be carefully examined, especially as to the quantity passed each day. The first sign of its diminution should be the signal for the administration of diuretics. The following prescription has been recommended for this purpose:

℞ Pulv. scillæ,
Pulv. digitalis, āā gr. xij;
Hydrarg. chlor. mitis, gr. ij.
Misce et fiant pilulæ No. xii.
Sig.—One pill three or four times a day.

The taking of large amounts of liquid should be insisted on, and the urine should be examined for the changes that indicate the development of nephritis. Albumen will usually be found present, at least in small quantities, whenever the icterus is marked. A certain number of casts will also be found; but as Litten has shown that occasional hyaline casts may be found in normal urine if carefully looked for after centrifugation, not much significance should be attached to their presence unless they are granular and abundant.

A serious condition, known as cholæmia, sometimes develops. It somewhat resembles uræmia. The prodromal symptoms are usually headache, slight drowsiness, a sense of fatigue, with lapse of memory and occasionally of consciousness; coma finally develops, and usually closes the scene. This condition was formerly attributed to the presence of biliary products in the circulation, but it is now regarded as a consequence of altered metabolism in the liver and in the intestines, and of a uræmic element due to a certain amount of attendant nephritis. When it develops, stimulants should be used freely,—ammonia, whiskey, a mustard plaster to the back of the neck, an ice-bag to the head,—and such remedies as benzoic acid and camphor are highly recommended by European authorities. Eichhorst advises the following prescription:

℞ Acidi benzoici, ʒiss;
Camphoræ, gr. xij;
Sacchari, ʒij.
Misce et divide in chartas No. xii.
Sig.—One powder every two or three hours.

An annoying symptom that often develops during the course of icterus is a sense of painful pressure in the hepatic region. This is due to an irritative hyperæmia of the liver, caused by the backing up of the bile. It can be relieved either by the application of leeches in this region, or perhaps still better near the anus, as it is here that the portal and the general circulation are connected. A

direct relief of the hyperæmia of the liver may sometimes be very rapidly effected by this expedient.

The duration of a case of icterus is very hard to foretell. It may last from three to five days to as many weeks; it may persist for months, and even for years. The first sign that the termination of the case is at hand is the reappearance of bile in the stools. When this is noted it can be announced with confidence that the icterus will soon abate. It must be remembered that the salts of bismuth cause a dark discoloration of the stools which should not be mistaken for bile stain. In the same way, calomel causes a green discoloration, which has not infrequently been attributed to biliary coloring-matter, and which has given calomel a somewhat undeserved reputation as a cholagogue.

THE TREATMENT OF ERYSIPELAS OF THE FACE.

CLINICAL LECTURE DELIVERED AT THE HÔTEL DIEU.

BY G. DIEULAFOY, M.D.,

Clinical Professor in the Paris Faculty of Medicine.

GENTLEMEN,—Erysipelas is such a common complaint that you may feel some surprise that I should choose it as a subject for my lecture; but I propose to discuss a recent innovation in its treatment that will, I am sure, justify the choice of such an every-day topic.

Erysipelas of the face is an exanthematic complaint which appears to be of microbian origin; in the opinion of Cornil and Babès the micro-organism that causes it is the streptococcus.

This disorder may result from trauma, and appear as a complication of any wound of the face; in its other forms it seems to occur spontaneously, without any lesion of the tissues that we are aware of, and is then known as medical erysipelas, in contradistinction to the first-mentioned form, which is called surgical erysipelas. In some cases erysipelas of the face is only a manifestation of erysipelas of the throat; in others still it occurs at fixed and regular intervals, as with certain women at the menstrual period, in which circumstances it is manifestly of internal origin.

The predisposing causes of erysipelas of the face are the same as those of erysipelas in general, and it will hardly be necessary for me to go into them in detail, as they are familiar to you all; I will merely say that grown persons seem specially liable to erysipelas of the face, while new-born infants are particularly exposed to erysipelas of the umbilicus. Finally, cachectic, diabetic, alcoholic, and nephritic patients are more subject to this disorder than persons in a fair condition of health.

Erysipelas usually commences on the face, and from there spreads gradually to the scalp; the reverse of this order, progression from the scalp to the face, is much less commonly observed. Gri-

solle's opinion was that erysipelas of the scalp is only rarely primary, except, of course, in the numerous cases in which it is of a traumatic origin and which do not concern us now.

The common variety that begins on the face starts almost invariably from the nose, forehead, or cheek, and spreads from these foci more or less rapidly and extensively to the rest of the face and the scalp. In cases where the point of origin is on the median line of the face, the spread of the erysipelatous patch is usually symmetric on either side.

We need not spend any time in discussing the nature of the lesions in this disorder, as the pathological anatomy of erysipelas of the face is very simple: the vessels of the skin are injected, and there is inflammation of the subcutaneous cellular tissue, with penetration of pus in some instances into the veins and lymphatic ducts; finally, when the form of the disorder is unusually severe, the blood itself is affected. Passing to the study of the symptoms of this complaint, we find that an outbreak of erysipelas of the face is announced by rigors, headache, nausea or vomiting, and by a sudden and abrupt rise of temperature, which often reaches 104° F. in a very brief space of time; it is usual to find, together with the foregoing symptoms, that the lymphatic ganglia in the submaxillary and lateral cervical regions swell and become painful. Although this condition of the lymphatic centres precedes the visible eruption of the erysipelatous patch, it is naturally a consequence of the infective process.

Soon afterwards the red patch characteristic of the disorder appears, and, beginning at the base of the nose, or at some other point on the face, invades progressively the remainder, either following an irregular course, or spreading symmetrically and occupying identical regions on either side. This inflamed patch may subside in the region first affected, as it gradually extends to other portions of the face, or else it remains in the same acute stage in all parts of its extent, in which case the patient becomes absolutely unrecognizable. The eyelids, which are enormous, completely cover the eyes; the face, tumified and distorted, loses all its outlines; the lips are greatly swollen, and the mouth remains open; the nose becomes misshapen, and the nostrils dry; all of these parts are painful on pressure.

The color of the patch, which may vary in intensity from pink

to scarlet, or even purple, and which disappears for the time being on pressure, is limited by a manifest terrace-like edge that is perceptible both to sight and to touch. Beyond this edge the skin retains its normal appearance in every way. In some cases the integument over the erysipelatous patch is very tightly drawn and shiny, while phlyctenæ appear over the entire area affected.

When the patch extends into the scalp, the redness cannot be perceived on account of the presence of the hair; at any rate, it is very moderate if not altogether absent. The only symptoms present in that region are pain on pressure, and œdema which can be perceived by the depression left after steady pressure by any pointed implement or by the finger.

In cases where the outbreak on the face is consecutive to erysipelas of the throat or pharynx, the mucous membrane with which these regions are lined is intensely red and shiny, and the local pain is very severe.

The general symptoms accompanying erysipelas of the face are sometimes quite violent; the high fever, mentioned above, is often attended by nervous manifestations, great agitation, and particularly delirium.

Such, gentlemen, are, briefly, the leading points concerning this disorder that I merely wished to recall to your memory before discussing the question of therapeutics, more particularly the use in this disorder of hypodermic injections of antistreptococcic serum.

The treatment of erysipelas of the face has hitherto been absolutely empiric, and, I must confess, of no efficacy. The many topic applications recommended for checking the spread of the disorder, —collodion, iodine, solution of nitrate of silver, mercurial ointment, solution of corrosive sublimate, etc.—have absolutely no value. All that we can claim is that the application to the inflamed surface of cloths dipped in some soothing decoction,—as of linseed, poppy-flowers, or elder-flowers,—and renewed very frequently, affords a certain amount of relief to the patient from the subjective sensations caused by the patch. The remainder of our therapeutic efforts were purely symptomatic: emetics and saline laxatives for the disordered condition of the digestive tract; opium (one or two grains), the bromides, chloral (fifteen to thirty grains), and quieting draughts when there was agitation or delirium; tonics, cinchona wine or extract and champagne when the adynamic symptoms predominated.

There is no question that the remedies just mentioned were of use when their exhibition was indicated, but they cannot be regarded as in any way curative of the disease. The rational treatment of this complaint, which checks its progress and cures it, is the hypodermic injection of antistreptococcic serum, proposed not long ago by M. Marmorek, of the Pasteur Institute.

This treatment, which is analogous to the treatment of diphtheria by injections of antitoxin, requires to be described somewhat in detail, owing to the steadily increasing importance of serotherapy in general.

In the preparation of this serum the first difficulty to overcome is to obtain in sufficient quantities a streptococcic virus sufficiently active and capable of retaining its virulence. We know from our studies of the streptococcus how to increase its virulence artificially; this is done by passing it from one culture-medium to another and from animal to animal, a series of cultures being thus obtained ranging in potency from those that are comparatively harmless to preparations of extreme virulence. An animal, a horse or an ass, is treated for a long time with these cultures in succession, until the most virulent has been administered; if, some time after that, his serum be taken, it is found to protect animals against the action of the most virulent streptococci.

In this way an antitoxin is obtained that promises to be of the greatest service in medicine. Just reflect for a moment on the frequency of streptococcic disorders in human pathology. Whether this micro-organism acts alone, as in erysipelas, puerperal infection, and in the different forms of inflammation, or whether it acts in association with other germs, as in diphtheria, sore throat, scarlatina, influenza, bronchopneumonia, or tuberculosis; whether it is itself a microbe of great virulence, or whether its virulence is increased by the microbes with which it leagues itself, there is no possible doubt that the streptococcus is an agent of the first importance in our nosology. If, therefore, an antitoxin has been found with which, either in its present form or in some future modification, we can cure, or even control, the onslaughts of this enemy, an advance will have been made in therapeutics the value of which it is hardly possible to over-estimate.

Of one thing we are certain, however, and that is, the efficacy of this serum in the treatment of erysipelas of the face.

Erysipelas is not a disease with a high death-rate. Still, according to statistics published by Chantemesse and based on his experience in the hospital devoted specially to erysipelas, this death-rate with our former methods of treatment was not less, and probably more, than five per cent. With the serum treatment this has been noticeably lessened, and not the death-rate only but the duration of the disease as well. In many cases both general and local symptoms are stopped short in their evolution.

The method of administering the serum and the effect it produces are as follows: An injection of ten, fifteen, or twenty cubic centimetres of serum is made under the skin of the abdomen with the usual antiseptic precautions. The complaint is thereby very rapidly modified; a few hours after the injection the patient feels better, the pain and the disagreeable sensation of burning and tension decrease, while in many cases the temperature begins to fall in six or eight hours and becomes normal in twenty-four. The redness and swelling of the patch lessen noticeably in from twelve to twenty-four hours, and desquamation has been observed to commence four or five hours after the injection. In cases treated in this way the suppurative forms that used to be so common in erysipelas are rarely encountered.

When it is found that a single injection of from fifteen to twenty cubic centimetres does not produce the amelioration we have a right to expect from it, a second and a third injection should be made on the next and subsequent days.

Patients treated by the serum show, in some cases, manifestations of erythema, with or without fever, which occasionally resembles purpura. These symptoms, which are in no way alarming, will undoubtedly disappear with a more perfect understanding of the preparation of the antitoxin. The horses that produce the serum should not be bled for four or five weeks after the last inoculation; during the weeks that immediately follow this inoculation the serum is still toxic, although it no longer contains streptococci.

The abscesses that in some cases follow these injections, and that have been found to contain streptococci, do not appear if the spot at which the injections are made is covered with iodoform collodion. These abscesses are due to auto-infection, and not to the antitoxic serum, which contains no streptococci.

Persons who are subject to recurring erysipelas, and women who are liable to it at their menstrual periods, might derive benefit from this treatment applied as a preventive measure.

The antitoxin treatment of erysipelas of the face may be regarded as the most rational and efficacious one we now have in our possession. The earlier in the course of the disease the injection is made, the better the effect that you will obtain from it. Thanks to this discovery, the death-rate in this disease has decreased, its duration has been shortened, the patients' suffering has been lightened, and the complications it produces can be avoided to a large degree. It is therefore entitled to take a prominent place in your therapeutic endeavors.

TREATMENT OF THE NIGHT-SWEATS OF PHTHISIS BY THE ADMINISTRATION OF SODIUM TELLURATE.

CLINICAL LECTURE DELIVERED AT THE LAENNEC HOSPITAL, PARIS.

BY ERNEST BARIE, M.D.,

Assistant Professor in the Paris Faculty of Medicine.

Gentlemen,—In the course of your practice you will meet with nothing more frequently in cases of pulmonary tuberculosis than profuse and repeated night-sweats, particularly in the advanced stages. The persistence of this morbid manifestation constitutes one of the most distressing features of this disease, and increases noticeably the patient's weakness.

It is customary to give a full description of this symptom in the text-books, and to it the term "night-sweats" has been applied; although, strictly speaking, this designation is inexact, as such "sweats" may occur as often in the daytime. This profuse perspiration comes on at any period of the twenty-four hours, provided the condition of its production exists, namely, *sleep*. You will then, of course, perceive that the reason they are called night-sweats is that the great majority of people sleep during the night-time. Still, those whose occupation in life compels them to sleep during the day have the perspiration during the period of rest in bed.

The patients find themselves bathed in perspiration when they awake, and this perspiration may be partial or general; it occurs with special frequency on the chest, neck, back, palms of the hands, face, and especially the forehead. Other patients complain that perspiration begins as soon as they commence to fall asleep, and becomes a most distressing source of insomnia; and this all the more owing to the fact that, unlike the physiological state of perspiration occurring in a condition of health as a result of heat, rapid walking, or severe manual labor, and followed by a sensation of coolness and euphoria, the sweats of tuberculosis are accompanied by

a general feeling of discomfort with unbearable warmth at the surface of the body, which is sometimes followed by an equally unpleasant impression of cold, due to the evaporation of the perspiration.

The amount of this perspiration is sometimes surprising, and in the very profuse cases you will note an eruption of sudamina of extreme confluence; in such cases the sweat seems fairly to trickle off the patient, whose night-dress, sheets, and pillow-case may be soaked through. This perspiration does not possess the peculiar sourish smell that we observe in cases of acute polyarticular rheumatism, but has rather the odor of fatty acids. From a chemical point of view, it contains mineral elements, such as the chlorides of sodium and potassium, urea, non-nitrogenous substances, chiefly fatty acids (butyric, formic, and acetic acids), solid matter, and water in large proportions, amounting to between 978 and 995 parts per thousand, approximately.

The phthisical night-sweat is not exclusively connected with the pyretic condition so common among these patients, or, at any rate, has no fixed relation to it; thus a consumptive whose temperature may be running very high may perspire only to a relatively moderate degree, whereas another whose fever is scarcely noticeable will have the most profuse perspiration. The celebrated physician Louis expressed this fact of every-day observation in the following concise and typical phrase: "Phthisical patients may have attacks of fever without perspiring, and can perspire without any rise of temperature." Another fact that shows plainly that there is no absolute connection between the pyretic condition of these patients and the abundance of their perspiration is that in acute miliary tuberculosis, in which there is often hyperpyrexia, excessive perspiration is probably less frequent, and at any rate less profuse, than in the chronic phthisical condition with only moderate rises of temperature. Furthermore, in the terminal period of this disease, at the approach of death, it is usual to see the patients bathed in perspiration while their temperature is only slightly above the normal, or it may even fall for a short period below the normal.

So far in this lecture you will have observed that I have almost always made use, in speaking of this form of perspiration, of the term consumptive in preference to that of tubercular patients; the reason for this is that night-sweats occur most usually in the ad-

vanced stages of tuberculosis, when the patients have been reduced to the cachectic condition answering to the state our predecessors designated specially by the term phthisis,—that is to say, a condition of extreme weakness and utter exhaustion of all bodily force produced by a consumptive disorder. Still, here again it does not do to be absolute, as night-sweats are also observed in the second stage of tuberculosis, where, if we are to believe Louis's experience in the matter, their average frequency is about ninety per cent. It may even happen that night-sweats occur in the first stage of the disease, disappearing when it reaches its height, to reappear again during the closing scenes of the tragedy.

The pathogeny of night-sweats in phthisical patients has not been made clear. Some of the early writers saw nothing more in this symptom than a simple phenomenon of compensation alternating with the diarrhœa that is so common in tuberculosis; but the frequent coincidence of these two morbid manifestations, particularly in the advanced stages of the disease, renders this hypothesis absolutely undefensible. Other writers have put forward the idea that owing to the diminution of the respiratory area in a seriously damaged lung there is produced an accumulation of carbonic acid gas in the blood, with signs of asphyxia, against which, in its effort to survive, the system resorts to this excess of cutaneous secretion. Lauder Brunton adopted a very similar theory, and thought that the excess of carbonic acid gas present in the blood overstimulated the nervous centres that preside over the sudoral secretion. Still other writers, finally, attribute the excessive perspiration to disturbance in the domain of the sympathetic system.

These various theories are, one and all, very difficult to maintain; and nowadays the majority of authorities are agreed in looking on the night-sweats of consumptive patients as produced by the toxins thrown off by the bacilli in the tubercular foci; according to this perception of the case, these toxic products impregnate the whole system, and by reacting on the centres of secretion stimulate that function to excess.

Whatever the reality may be as regards this pathogeny, the night-sweats of tuberculosis are one of the most distressing symptoms with which these unhappy patients have to battle; they disturb their rest, even when they do not prevent it entirely, and are

a cause of marked decrease in strength. It is, therefore, manifestly the physician's duty to put forth every effort to check this perspiration; and there are quite a number of remedies with which this can be accomplished. You will find them mentioned in your treatises on therapeutics. At present I shall merely mention the most reliable ones, with their doses, laying particular stress on a relatively recent addition to the list with which many of you are probably not familiar.

I think you may strike the *acetate of lead* out of this list of drugs; its action on this symptom has never been very manifest, while, on the other hand, it has in some cases produced serious attacks of lead-poisoning.

The *white agaric* has been known and administered for a long while; it is a good remedy; but, unfortunately, its effect is not lasting. It must not be given in large doses, as it then acts as a drastic; four or five grains of the powder in a wafer are enough, —and it may be preferable to divide this dose into two parts, to be taken separately during the evening before retiring.

The *sulphate of atropine* is an alkaloid that is far more efficacious than the white agaric; it rapidly moderates the activity of the sweat-glands, but this moderating action affects the salivary glands of the mouth and throat as well, causing a very unpleasant condition of redness and dryness. Finally, its action on the power of accommodation disturbs the sight in certain patients. When administered with care, and when its effect is closely watched, this remedy is an excellent one for the night-sweats of consumptives, in whom it lessens at the same time the bronchial secretions and diminishes the attacks of spasmodic coughing, of which many of them complain so bitterly. You must, however, always bear in mind the toxic properties of belladonna and handle this remedy with prudence; it is usually given in pellets containing one-quarter of a milligramme ($\frac{1}{260}$ of a grain), of which from one to three should be given during the evening with an interval of two hours between each pellet.

Ergot has also been recommended on account of its action on the unstriated fibres of the vessels, which it causes to contract; it is thought thus to act directly on the vessels of the sweat-glands or on the glands themselves, of which the larger ones possess in their structure longitudinal unstriated fibres.

Camphoric acid has a rather slow effect, and can be given either in wafers or in alcoholic solution; the former seems preferable,—that is, in wafers containing fifteen grains, of which two or three can be taken *per diem*.

The *tribasic phosphate of lime* also has been warmly recommended, in doses of sixty grains a day; this amount should be taken in the middle of the day in two equal parts with an interval of fifteen to thirty minutes between them.

Tannin and *oxide of zinc* also are used, the first in doses of five to eight grains, the second in an average dose of eight grains, and the fluid extract of *Hydrastis canadensis* can be given, thirty drops at bedtime; but these are not remedies that can be relied on and their action is not lasting.

Such, on the other hand, is not the case with *sodium tellurate*, to which I wish particularly to call your attention. Neusser, of Vienna, was the first to test the therapeutic effect of the tellurates (1890). He first used the tellurate of potassium, and gave it in fifty-one cases of pulmonary tuberculosis in doses of one-third of a grain without any bad symptoms or even signs of intolerance. He noted, however, that the sweats were checked in all his patients, and reported that no undesirable phenomena occurred unless the remedy was given in doses of a grain *per diem* and for several successive days.

Very soon afterwards (1891), Combemale, of Lille, took up the question again; but he used the tellurate of sodium in the place of the same salt of potassium on account of the lesser degree of toxicity of the sodium salts as compared with those of potassium. Since that date this remedy has little by little entered into the domain of the treatment of pulmonary tuberculosis; and I think that after the above-mentioned writers I was one of the first to test and then make known this treatment. In 1896 one of my students, Dr. Jaquet, based the thesis he wrote for his degree on the close study of twenty cases of tuberculosis with profuse sweats then in my wards at Tenon Hospital.

Sodium tellurate is a whitish powder, soluble in water and alcohol; it is eliminated from the system through the kidneys and lungs, and this pulmonary exhalation is shown by the persistent smell of garlic that it gives to the breath. Neusser claims that this salt has a peculiar antiseptic effect, that it is even a powerful

bacterial agent capable of modifying the action of the toxins secreted by the infectious agent, and that it is in this way that it prevents the night-sweats of consumption. Without going any farther into this point, and relying wholly on clinical observation, there is no denying the fact that sodium tellurate is one of the most valuable agents in our possession for the treatment of phthisical night-sweats. It appears, on the contrary, to have no effect on normal perspiration.

It has no toxic effect. During the last five years I have given it to a large number of consumptives, and in no instance with any serious inconvenience. Others have reported colics, or slight diarrhœa, and this may be possible when the therapeutic dose is exceeded; but the only real drawback to its use is the smell of garlic it gives to the breath. This, however, is a matter of minor importance, and should deter neither physician nor patient.

The dose of this remedy is one-third to one-half a grain *per diem* on an average, for three or four consecutive days. If the sweats are not modified, it is possible, after testing the ground, to give five-sixths of a grain divided into two equal doses; this dose may be well tolerated, but I do not advise more than that. Still, in exceptional cases I have increased the dose, but in these cases I obtained no success, the patients being apparently refractory to the drug. Such instances are indeed exceptional; while the majority of patients suffering from profuse sweats derive from this preparation undeniable and lasting relief. When the sweats reappear it is usually after a long period of cessation, but no hesitation should be felt in resuming the use of the drug at the usual dose.

The form in which the sodium tellurate is to be given is, I think, indifferent; you may use the alcoholic solution or the pill form,—I myself usually prescribe it in pills.

ASTHMA.

BY AUGUSTUS A. ESHNER, M.D.,

Professor of Clinical Medicine in the Philadelphia Polyclinic; Physician to the Philadelphia Hospital; Assistant Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases, etc.

Difficulty in breathing may obviously arise from a variety of causes, and most commonly from disease of the lungs or pleuræ, of the heart, and of the kidneys. Functional derangement of the same organs and disease or compression of the upper air-passages also may cause dyspnœa. The conditions thus resulting must, however, not be confounded with asthma, although the names cardiac asthma and renal asthma have been employed; they may with propriety be designated asthmatoid. True asthma may be looked upon as a respiratory neurosis of paroxysmal character attended with dyspnœa, especially of inspiratory type. Whether the disorder is dependent upon spasm of the bronchial muscular apparatus or due to swelling of the bronchial mucous membrane has not been finally determined, but it is not improbable that both sets of influences are operative, perhaps in different cases, possibly in the same case at different stages. Vasomotor spasm and vasomotor paresis respectively may also be contributing factors. It may be that primary tonic muscular contraction is followed by secondary muscular relaxation. Whatever the explanation, relaxants usually do good in the acute stage, and sometimes also in the intervals between attacks, when tonics likewise are often of service. The view has, further, been propounded that the asthmatic paroxysm is dependent upon an inflammatory or an exudative process involving the smallest branches of the bronchial tree, an exudative bronchiolitis.

As with other neurotic affections, the predisposition to asthma may be inherited. The disease may set in early and continue throughout a long life. Males are more commonly affected than females. Atmospheric conditions play an important *rôle* in the excitation of attacks. Reflex disturbances, especially of nasal origin,

are by some considered potent etiologic factors. No doubt the toxic results of derangements of metabolic equilibrium are responsible in some, if not in many, cases.

The paroxysms recur with varying frequency. They may set in abruptly or gradually. Often they begin at night. They are characterized by dyspnœa, sometimes of extreme intensity, and attended with an appearance of the utmost anguish. The sense of the need of air may be most pronounced. There is little respiratory movement, the chest being fixed in inspiration, and there may be some cyanosis.

On physical examination the chest is found almost immobile, and the breathing labored. The percussion-note is little, if at all, altered, and fine, high-pitched, musical râles are audible, together with snoring sounds. The attack is of variable duration. It may terminate in a few minutes or a few hours, with free or copious expectoration; and it may be repeated on several nights in succession, amelioration taking place, or perfect relief being afforded, during the day. The sputum may be viscid and translucent or opaque; in it may be found characteristic spiral structures or elongated octahedral crystals and eosinophilous leucocytes. The blood also contains an increased number of eosinophile cells. If the attacks be repeated frequently, bronchitis often becomes an associated disorder, and emphysema is likely to be a sequel; and each adds its own special features.

The diagnosis of a typical attack of asthma is not difficult. Care must, however, be exercised to discriminate between the deuteropathic asthmatoid states and the protopathic disorder. The clinical likeness may be exceedingly great, and the differentiation will depend upon the history, especially with regard to previous attacks, and upon the presence or absence of nasal or of cardiac or of renal disease.

For the paroxysm the most effective treatment consists in the hypodermic administration of morphine in dose sufficient to induce physiologic effects,—from one-eighth to one-half grain. With it may be conjoined atropine (gr. $\frac{1}{200}$), hyoscine (gr. $\frac{1}{150}$), or cocaine (gr. $\frac{1}{4}$), and also strychnine (gr. $\frac{1}{30}$). The injection may be repeated cautiously once or oftener, at intervals of an hour or two. I have reported a case of asthma in which a single hypodermic injection of morphine sulphate (gr. $\frac{1}{8}$), strychnine sulphate (gr. $\frac{1}{60}$), and hyoscine hydrobromate (gr. $\frac{1}{100}$) was followed by alarming

symptoms of hyoscine intoxication.[1] Inhalations of amyl nitrite or of chloroform-vapor sometimes afford marked relief. Good results are yielded also by inhalation of the fumes yielded by burning the leaves of belladonna, hyoscyamus, or stramonium, with or without potassium nitrate. Nitroglycerin (gr. $\frac{1}{100}$), spirit of chloroform (ʒss–ʒi), and compound spirit of ether (ʒi) are useful in some cases. Paraldehyde in drachm doses, or chloral in doses of from fifteen to twenty grains, may be employed with hope of benefit. Counterirritation of the chest with mustard or turpentine, or dry cupping, may mitigate the severity of a paroxysm. An emetic dose of ipecac or of apomorphine may hasten the termination of an attack.

In a case of some standing seen recently most gratifying effects followed the employment of the following somewhat comprehensive formula:

℞ Hyoscine hydrobromate, gr. ½;
Strychnine sulphate, gr. ¾;
Morphine sulphate, gr. vi;
Sodium bromide, ʒvi;
Solution of potassium arsenite, fʒii;
Tincture of digitalis, fʒiv;
Compound infusion of gentian, q.s. ad fʒvi.—M.

Dose.—Two teaspoonfuls every three hours.

The danger of the formation of the opium habit in these cases should never be lost sight of.

In another case, in which the foregoing prescription was used without appreciable effect, the symptoms subsided following the administration of eight grains of quinine sulphate and one-fourth grain of codeine. In a third case, complicated by pleurisy with effusion, speedy relief succeeded upon the administration of the following formula, after other measures had failed:

℞ Fluid extract of grindelia,
Fluid extract of lobelia,
Fluid extract of quebracho, āā fʒi.—M.

Dose.—A teaspoonful thrice daily.

In the intervals between attacks accessible causative influences should, so far as possible, be removed, nasal and bronchial disease

[1] Therapeutic Gazette, October 15, 1897.

treated, digestive and metabolic disturbances corrected, and special attention directed to the restoration and maintenance of general physiologic equilibrium. The functions of the skin, lungs, kidneys, and bowels should be kept active by judicious exercise, bathing, diet, and rest. Massage and hydrotherapy are often of especial service, and inhalations of compressed air and other forms of pulmonary gymnastics are to be recommended in some cases. Among medicinal agents that have proved most useful, singly or in varying combination, are iodine and iodides, arsenic, strychnine, ergot, quinine, nitroglycerin and nitrites, lobelia, grindelia, quebracho, and cod-liver oil. Iodipin, a ten per cent. combination of iodine with sesame-oil, has been employed successfully in doses of a fluidrachm twice or thrice daily. Picrotoxin may be administered instead of strychnine, and adrenal extract may take the place of either. Small doses of atropine or of hyoscine, or of tincture of belladonna or of hyoscyamus, may prove useful.

Medicine

CHRONIC INDURATIVE NEPHRITIS AND THE SIGNIFICANCE OF ALBUMINURIA.

CLINICAL LECTURE DELIVERED AT THE CHARITÉ HOSPITAL, BERLIN, GERMANY.

BY PROFESSOR H. SENATOR,

Director of the Third Medical Clinic and Professor of Special Pathology and Therapy in the University of Berlin.

GENTLEMEN,—I have had our patient of the last clinic brought in for a few minutes at the beginning of this one to impress upon you the contrast that is to be found in cases of chronic nephritis, —chronic Bright's disease. Later, I shall have another patient brought in, and you will see the striking external differences that may exist between patients suffering from a malady bearing the same name. These are extreme cases, and you will find in practice all shades of variation between the extremely cyanotic form we have here and the extremely pale one that we shall see in a few minutes.

Of course, besides these cases, that present at least some characteristic symptoms or other evidences of chronic disease, there occur not a few cases of chronic nephritis that present absolutely no symptoms of disease, the patients in question producing the impression of perfect health; only the examination of the urine brings to light the insidious affection that is present. You have often seen such patients in the dispensary service, and I have tried each time to impress upon you the necessity, in every case, of making an examination of the urine for albumen, and in suspicious cases of inspecting that collected at different times during the day, and especially not to depend on the examination of the morning urine alone, for it often happens that, while the urine secreted during the

night contains no albumen, that passed during the day contains a considerable amount. Ordinarily, unless special instructions are given, it is the night urine that patients bring with them to the doctor.

For all these varied conditions of kidney affection only one symptom is common, the albuminuria, without which we can diagnose neither acute nor chronic nephritis, no matter what its form. At most, we can suggest the possibility of a nephritis when we find other symptoms, and for the moment no albumen is present in the urine. But I shall return to this question of albuminuria and nephritis in a few minutes, after I have shown you the patients whose very different symptoms have suggested this talk upon chronic nephritis.

This patient, a man of forty-four, had when younger a number of characteristic attacks of gout. As the result of the gouty diathesis there has developed the chronic nephritis that is a frequent consequence of it. The disease has in this case run its typical course. Gout causes, as is well known, that form of chronic nephritis in which the interstitial tissue is principally and perhaps primarily affected. No special tissue of an organ, however, can be affected without the others being involved, and so the parenchyma, the glomeruli, and the urinary tubules gradually disappear, the interstitial tissue contracts, as does all connective tissue of inflammatory origin, and the so-called contracted kidney results.

Gout is so common a cause of contracted kidney in England that this form is sometimes called "gouty kidney." We meet with it not rarely here in Germany.

The abnormal condition of the blood that we call gout may affect the kidneys in two ways. First, the toxic material in the circulation may act directly as an irritant upon the kidney substance, parenchyma as well as interstitial tissue, and thus produce a primary diffuse chronic nephritis. Secondly, the abnormal blood constituents may act directly upon the arteries, setting up an affection of them that we designate as arteriosclerosis. When this arteriosclerotic process affects the arteries of the kidney, there results, because of the lowered nutritional conditions, a contraction of the glomeruli and the urinary tubules, and inflammatory processes in the interstitial tissue follow, so that finally there is, just as in the first case, a contraction of the kidney, the origin of which, how-

ever, was an arteriosclerosis, and so we have the arteriosclerotic contracted kidney, or kidney sclerosis.

Naturally, in many cases of gout, the disease acts both directly and indirectly upon the kidney, so that both forms of kidney induration, the primary interstitial and the secondary arteriosclerotic, run their course side by side, and it is practically impossible absolutely to differentiate them from each other.

There are other causes besides gout that may produce chronic indurative nephritis or arteriosclerosis, or both together. The most important of them are the abuse of alcohol and chronic metallic poisoning, especially lead-poisoning. It is probable that syphilis, the abuse of tobacco, diabetes mellitus, plethora and polysarcia, and heredity, also, may play a *rôle* in the etiology of these conditions.

In this case the condition is complicated, as happens not infrequently, by the presence of obesity; and there is no doubt that the disturbed heart compensation is in a measure due to the fatty deposit around the heart, and to the hampering of its action which that involves. The external symptoms of the disease are mainly those from the heart. The color, especially that of the face, is deeply cyanotic; the lips in particular, the hands, feet, legs, and arms share this bluish-red color. Inspection shows that there is venous congestion of their capillaries. The legs are œdematous, most so farthest from the trunk, and the hands are also dropsical. The face, however, is perfectly free from œdema.

The dropsy is here evidently due to slowing of the circulation in the peripheral capillaries. We might think of a cardiac valvular lesion in the case, with loss of compensation, the albuminuria being an expression of the circulatory disturbance and venous congestion in the kidneys. One very significant phenomenon speaks against this,—the appearance of the urine. You can see that, despite the extreme cyanosis, it is pale in color and has a low specific gravity, 1008, while the amount passed in twenty-four hours is not as small as it would be if the symptoms here were due to a cardiac lesion and disturbed circulatory compensation. Indeed, the amount of urine is often above the normal as in our case here, and it is not unusual for the patient to pass two litres, or quarts, in twenty-four hours. The urine, we may say in passing, gives only a small sediment, in which from time to time a few hyaline casts are to be found, though occasionally slightly granular casts are present.

This appearance of the urine and its amount are very important symptoms of chronic indurative nephritis, for during the course of indurative nephritis the hypertrophied heart finally begins to fail in its work, and then the symptoms are very like those that come with valvular failure of compensation and consequent renal congestion. These conditions of the urine, then, are significant only in a positive sense; that is to say, when in cardiac hypertrophy with cyanosis and dropsy the albuminous urine is pale and of low specific gravity, it is evidence of chronic indurative nephritis; but the opposite does not hold, for chronic indurative nephritis may, after cardiac insufficiency has developed, give the typical urine of renal congestion.

Let me show you another and, as you will at once see, a very different form of chronic nephritis.

This patient is forty, a locksmith; his mother died of cancer; his father, brothers, and sisters are alive and well. He was always perfectly healthy up to about six months ago. Then a tired feeling came over him, an intense disinclination to work, and he had some fever. He consulted a physician, who put him on an absolute milk diet. He improved and went to work again. Then symptoms of dyspnœa began to develop and disturbances of vision set in. When he was brought to the Charité he was completely amaurotic. Since then his dyspnœa has increased, until now we have a condition of dyspnœa maxima. You note that at every breath, which is rather a gasp than a respiration, there is a convulsive movement of the lower jaw. It is seldom seen in adults, though not uncommon in children, and is always a sign of intense dyspnœa. In rabbits it may commonly be observed even in the light forms of dyspnœa.

You may note, too, that all the accessory muscles of respiration are at work. His sternocleidomastoid muscles stand out in intense contraction, in order to give points of support for the action of the external thoracic muscles. The muscles of the shoulder-girdle are on the stretch for a like purpose, and the muscles of the upper thoracic region are brought into what is exaggerated action for a man, for in most men costal breathing is scarcely perceptible. There are long excursions of the larynx, the sternothyroid muscles being called into excessive action.

There is a striking contrast between the cyanosis of the last patient and the intense waxy paleness of this one. This pallor is

extreme, even in the mucous membranes. While the last patient was fat, this one is emaciated, though the emaciation of the legs is concealed by the presence of a marked œdema. When he first came to the hospital his face was puffy, but very pale. The puffiness has disappeared; the waxy pallor remains. Our first patient had all his senses perfectly. This one is somnolent, pays no attention to things that go on around him, and is not easily aroused. The œdema and the general dropsy would suggest at once either heart or kidney trouble; but where the patient is as pale as the present one the heart as a primary cause can be excluded at once. The kidneys alone, but not the heart alone, may produce as much œdema as this, and with it this pallor. Only one other condition can give œdema with pallid skin, and that is severe anæmia, or some serious disturbance of blood composition leading to hydræmic conditions.

The heart sounds are normal. The pulse is weak and easily compressible, but perfectly regular. There is often a difficulty in feeling the pulse of patients with as much œdema as here; and it is always well to press the œdema away before attempting to feel for the pulse, or the judgment will be warped by its seeming distance and indistinctness. The pulse may, however, be felt in the temporal artery or in some other location where there is no œdema; and it is always well to do this, so as to control the impression obtained from the radial when there is œdema around it.

We find an area of dulness here at the bases of both lungs, which we very naturally, under these circumstances, conclude to be due to serous effusions into the pleura, so-called hydrothorax. We find the abdomen extremely distended, though we do not find any tortuous, distended veins running over it, as so often occurs in ascites from congestion in the portal system. The navel is prominent, and the ordinary physical signs of fluid in the peritoneal cavity are easily demonstrated.

Vision is still seriously disturbed. On admission there was, as I have already told you, complete amaurosis, but some faint glimmer of sight has returned. Ophthalmoscopic examination shows the presence of advanced papilloretinitis.

Here is his urine. Putrefactive processes were at work in it before it was passed, so that even when fresh it has a striking ammoniacal odor and alkaline reaction. An ammoniacal smell and alkaline reaction develop in a very short time in highly albuminous

urine if it remains exposed to the air. As the significance of these changes is very different if they have developed outside or are already present in the urine while in the bladder, it is always well to take the most careful precautions to examine only absolutely fresh specimens for these signs.

In cases like this one, in spite of the most careful precautions and even though no catheter has been used, micro-organisms from the meatus, where they grow abundantly because of the albuminous contents of the urine, find their way into the bladder. The urethral flora generally are more plentiful in such cases, and eventually they reach the bladder and set up decomposition in the urine. Under the influence of these micro-organisms the urea takes up water and is converted into its isomer, the carbonate of ammonium. While in many of these cases catheterization is to blame for the introduction of micro-organisms into the bladder,—the most aseptic catheter being liable to take up urethral flora in its passage, and so be anything but aseptic when it reaches the bladder,—still, it is to be remembered that this intravesical decomposition of the urine may take place simply as the result of a cystitis where no catheter has been used, and sometimes develops when there is no cystitis present, as our patient here serves to show.

The presence of ammonia as a volatile alkali may be easily detected by the characteristic odor of the urine, or may be demonstrated chemically by holding over it a rod dipped in hydrochloric acid, when, as you see, the white, smoke-like fumes of chloride of ammonium become visible.

In testing such urine for albumen, it is necessary to add an excess of nitric acid, since a certain amount of the acid is taken up in neutralizing the alkali present, and the albumen may remain in solution.

The contrast between the urine of this case and that of the first one is very striking. In the first, the interstitial nephritis case, the urine was clear, light yellow in color, plentiful in amount (always nearly two litres, or quarts), of lowered specific gravity (usually 1008 or lower), with only slight amounts of albumen in it, and only a small sediment, in which casts were occasionally to be seen. Here we have a darker, cloudy urine, small in amount (usually less than five hundred cubic centimetres, or one pint, in twenty-four hours), of high specific gravity (1030 and above), with large

amounts of albumen, and, until the present intravesical urinary decomposition had set in, letting fall a copious sediment in which casts were numerous.

There is now not much sediment, casts are scarcely to be found, and those present are in a state of granular disintegration, but that is because they become disintegrated during the process of decomposition. Before the present alkaline fermentation set in they were found, as we have said, in abundance. This fact, the disintegration of casts when ammoniacal decomposition has taken place, must not be forgotten, for the presence of casts is always an important sign in deciding whether an affection of the kidneys really exists or not. As the highly albuminous urine of chronic nephritis is especially liable to this decomposition, the necessity for its reception and preservation in perfectly clean vessels, and for its examination as soon as possible after its passage, becomes evident.

As a result of our consideration of this case and its symptoms, we conclude that we have here a chronic parenchymatous nephritis in the process of conversion into so-called contracted kidney.

I have said that we cannot diagnose nephritis without albuminuria, but it does not follow from this that every albuminuria means nephritis. It was the custom some time ago to say simply, where there is albuminuria there is Bright's disease; but we have learned that there is a series of conditions, not inflammatory in character, in which albumen appears in the urine. More than this, we have found that albuminuria may occur where there is no pathological condition demonstrable either in the kidneys or in any other organ, so that it may be considered as a genuine physiological albuminuria.

The same thing holds for the excretion of albumen in the urine as for sugar and many other substances whose presence in the urine we formerly regarded as pathological, but that we now know may under certain conditions, physiological or pathological, occur in the urine in larger amounts than usual, and so become easily demonstrable. This holds for such substances as oxalic acid, hippuric acid, indican, and the like. When we speak of glycosuria, oxaluria, indicanuria, etc., we do not mean that these substances never occur in normal urine, but that they do not occur in any considerable amounts. It is quite the same for albuminuria. It has been shown beyond all doubt, now, that albumen occurs normally in the urine of

men and animals, but in such small amount that it may be ignored in practice. We speak of albuminuria only when it occurs in larger amounts and may be demonstrated by the ordinary tests, just as we neglect the minimal amount of sugar that occurs in normal urine and speak of glycosuria when the ordinary tests for sugar are positive. Just as every glycosuria is not pathological and should not be taken as a symptom of diabetes mellitus, so every albuminuria is not a sign of disease and should not be considered as indicative of nephritis.

Just as after an excessive meal of carbohydrates alimentary glycosuria may be noted, so after a meal in which large quantities of albumen have been consumed alimentary albuminuria may occasionally occur. Certain nervous disturbances and psychic conditions may give rise to glycosuria, and certain of the neuroses,—mental overwork, great emotion, fright, anxiety, and the like,—may cause albuminuria. There the comparison ends. While muscular exertion causes the disappearance of sugar from the urine, there is no doubt, now, that in perfectly healthy individuals, after unusual and severe exercise, especially where the lower extremities are brought much into play, as in soldiers after a forced march, in tourists after a long pedestrian excursion, in mountain climbers, in bicyclists,—and recent observations as to this have been very frequent,—albumen is found in the urine. The same thing holds true for exposure to cold and for cold bathing.

All these causal factors—a plentiful meal, an exhausting march or ride, a cold bath—are within the limits of the physiological. The albuminuria disappears as soon as the occasion that has given rise to it ceases to favor its occurrence; no sequelæ are noted, no trace of the condition remains, so that we cannot think that there was an inflammatory or any other pathological condition in the kidneys. Under such circumstances it is evident that we may speak of physiological albuminuria.

But the diagnosis of the condition must be made with the greatest care and circumspectness. The personal history of the individual in question must be well known in every respect, repeated examinations of the urine must have been made, one or more of the causes that I have mentioned for this physiological albuminuria must be present, the albuminuria must cease when this cause no longer exists, and, finally, there must be nothing else abnormal about

the urine. The occurrence of a single cast is sufficient, in my opinion, to preclude the idea of physiological albuminuria. Urinary casts occur only as the result of the shedding of the epithelium of the urinary tubules. They presuppose an irritation or a nutritional disturbance of the epithelium, and this, in its most favorable aspect, must be considered to have gotten beyond the bounds of what is physiological, and therefore must be considered as a warning of serious danger.

As a general rule, anything more than a small amount of albumen cannot be looked upon as physiological. For myself, I consider that more than 0.04 to 0.05 per cent. of albumen is at least suspicious, and indicative of a pathological condition.

After this statement of the question, I need scarcely add that there are no sharply defined limits between physiological and pathological albuminuria, just as there are none between alimentary glycosuria and diabetes mellitus. In every case a long period of observation is required, with the careful exclusion of all pathological factors, before we dare come to the conclusion that a physiological albuminuria is present, and in all doubtful cases the inclination must be rather to the acceptance of a pathological albuminuria.

But not even every pathological albuminuria signifies nephritis. Apart from all inflammation, it may be caused by circulatory disturbances,—*i.e.,* by venous congestion in the kidneys, or by degeneration of the renal parenchyma without inflammatory processes, especially by amyloid degeneration, more rarely by fatty degeneration. Since we are concerned to-day with chronic nephritis, I ignore entirely acute nephritis and the so-called febrile albuminuria.

A congestive albuminuria we diagnose almost entirely from the condition of the urine, which is small in quantity, darker, and more concentrated than normal. It often allows the precipitation of urates, and contains from time to time very variable quantities of albumen; on the other hand, it has no morphologic elements, or at most a few hyaline casts, and sometimes a leucocyte or two. We find in addition to all this, however, other symptoms of venous congestion, cyanosis, œdema of the most dependent parts, and finally some cause for the congestion, as a non-compensated valvular lesion, emphysema, or local disturbances of the renal circulation (ascites, tumors, etc.).

Amyloid disease of the kidneys is often not recognized. Its diagnosis is possible only when it is known that some of the causes that usually produce amyloid degeneration have been at work, and when, besides this, there are clinical evidences of amyloid change in other organs. The usual causes are chronic tuberculosis, long-enduring, active syphilis, chronic purulent processes, especially caries of bone, and the cachexias of such affections as carcinoma, rickets, or long-standing gout, though these last are comparatively rare as causes. It has been demonstrated recently that not only microbic pus, but also the artificial purulent secretion obtained as the result of the aseptic application of such irritants as turpentine, may, if the irritative process be kept up long enough, produce amyloid changes in internal organs.

Not the kidneys alone are affected, but we may have amyloid disease of the liver, the spleen, and the intestines. The examination of these organs, then, is always of importance for the diagnosis of amyloid disease. As the result of amyloid change in the liver, we find it regularly enlarged, not much, if any, changed in consistency, not painful, and there is usually no jaundice. About the same characteristics are found clinically in the amyloid spleen. In the intestines amyloid change gives rise to severe and obstinate diarrhœa, and when, as happens in rare cases, the mucous membrane of the stomach is affected by the amyloid degeneration, the hydrochloric acid reaction in the stomach may disappear, and the stomach contents may even have an alkaline reaction. The urine in amyloid degeneration of the kidneys may have very varied characteristics; at times it is scanty and dark colored, and again clear and plentiful, usually containing copious albumen. When it is clear,—*i.e.*, without sediment,—or contains at most a hyaline cast or two, and the other diagnostic factors that we have mentioned point to an amyloid condition, and when, besides, the patient is extremely pale and anasarca is present, we may make a diagnosis of amyloid disease with a certain amount of assurance, otherwise the diagnosis is only a matter of more or less probability.

On the other hand, amyloid disease of the kidneys may be found post mortem without its presence having been betrayed by a single symptom during life.

The difficulties of the diagnosis are rendered more insurmountable by the fact that at times a combination of amyloid disease and

inflammation occurs, the so-called amyloid nephritis, as is found in what has been named the large white kidney.

Fatty degeneration of the kidney may be surmised when certain causes of fatty degeneration are known to have been at work (as phosphorus-poisoning, severe anæmia, and the like), and when at the same time the urine is scanty, either pale or dark, and contains some albumen, some fatty granular matter, or fatty degenerated cells.

As regards chronic inflammatory affections of the kidney, chronic nephritis in its most literal sense, the various forms can be sharply differentiated neither anatomically nor clinically; but there occur certain clinical pictures which may be considered as types of special forms, though all of these affections terminate finally in the indurative, contracted form, if the patient is not carried off by some intercurrent disease. According to the origin and course of the pathological process, we may differentiate a primary and a secondary indurative or contracted kidney, and a third, the arteriosclerotic form, may be added.

As to origin, first, a chronic indurative nephritis not infrequently results from an acute nephritis. A child, for instance, develops acute nephritis after scarlet fever or influenza or diphtheria, or some one of the other infectious diseases. The first serious symptoms gradually disappear, the dropsy goes down, the urine becomes abundant and clear, the albuminuria grows less and less, until finally, some morning, none can be found, and then the child is pronounced well. Had not only the morning urine, but that passed in the course of the day been examined, albumen would still have been found, and the child would not have been allowed to get out of bed. Or perhaps the urine passed during the day is found free from albumen, but further urinary examinations are neglected after the child has been up and around for some time, and so it passses for perfectly healthy, until finally, by chance, albuminuria is found once more; or, after a year or two, all sorts of inexplicable symptoms develop,—headache, tired feeling, palpitation of the heart,—and then the urinary examination discloses polyuria, pollakiuria, especially at night, while the preliminary symptoms of cardiac hypertrophy become ever more and more noticeable, until, finally, the complete clinical picture of contracted kidney is developed.

When in the course of years one has had the opportunity to see

the disease gradually develop out of acute nephritis, he must regard the chronic nephritis as secondary; but very often the physician knows nothing of this origin. He may never have seen the patient before; or the patient, who has grown up in the meantime and has always considered himself perfectly healthy, remembers nothing of the infectious fever he had years before; in fact, the infection may have passed almost unnoticed, for it may have been only an ordinary angina. And so it is that when a physician finds a chronic nephritis, he is apt to speak of its having developed spontaneously, and to call it primary chronic nephritis because the original acute nephritis was overlooked.

It is, however, possible that chronic nephritis may be truly primary,—*i.e.*, originate without having been preceded by an acute nephritis. This form develops extremely slowly, without œdema and without noticeable symptoms of any kind, and may remain for years absolutely latent, there seeming to be no reason for an examination of the urine, and so the albuminuria remains undiscovered; or an examination of the urine may be undertaken, but, as so often happens, even in our day, it is only the morning urine that is examined, and in this no albumen may occur, though it might be easily found in the urine passed during the day. Let me insist on this important point, that the day urine often contains albumen when the night urine does not, while the opposite seldom occurs, and then only in consequence of unusual excitement and exertion during the night (coitus, masturbation, pollutions, etc.). Cases such as these may for a long time be regarded as physiological albuminuria, until after years, because of unsuitable treatment, the signs of contracted kidney develop, and polyuria, pollakiuria, with pale, clear urine, containing ever-varying amounts of albumen, cardiac hypertrophy, rigid arteries, and the like, manifest themselves, when, of course, the pathological nature of the albuminuria can no longer be doubted.

In a somewhat different way do indurative nephritis and contracted kidney develop from the so-called chronic parenchymatous nephritis. This last has, as is well known, a great similarity to acute nephritis, though the differentiation is not difficult because of the insidious beginning, the tedious course, and the fact that the cause for it can very seldom be found, and, as a consequence, it is usually traced back to a "cold."

This chronic parenchymatous nephritis very seldom ends in cure after it has once existed for some time; the much more usual termination is death, which comes from waterlogging of the tissues and interference with organic functions because of the dropsy, or from inflammatory processes, especially of the lungs, or from uræmia. At times, however, it takes on a more favorable course; after a year or two the dropsy disappears, the urine becomes more plentiful, paler, and clearer, the morphological sediment lessens in quantity; only the albuminuria persists. Then there gradually appear in the circulatory system changes which are characteristic of contracted kidney,—cardiac hypertrophy, high arterial tension, etc.; and we find that a secondary contracted kidney has developed, with which the patient may live on in passably good condition for years. This form of contracted kidney every one admits to be secondary, since the primary stadium (the parenchymatous nephritis) cannot well be missed, as happens so often in the other case.

Finally, the indurative nephritis and contracted kidney, as I have already said, may develop as the result of an arteriosclerosis, the arteriosclerotic condition being in older people senile arteriosclerosis, or in younger ones the result of some of the toxic causes we have mentioned. In these cases the changes in the circulatory system, the thickening and lengthening, with consequent tortuosity, of the arteries, the dilatation and hypertrophy of the heart, especially of the left ventricle, cardiac asthma, and angina pectoris become gradually noticeable; and then, in its subsequent progress, the changes in the urine, which becomes clearer and more abundant, with albuminuria at first only periodically and in small quantities, later on greater in amount and persistent. These cases show very early in their course the typical picture of contracted kidney as it was described by Traube.

I repeat that not every case presents as typical a picture as this. What we have described here is the end and result of the series of changes that take place. It develops at times sooner, at times later; not infrequently only after years reaching a characteristic stadium. Its development does not always take place under the eye of the doctor; the commencement of the disease is often unrecognized or the history unreliable and incomplete, so that we cannot always say what form of nephritis we have under observation, and must be satisfied, then, to call it chronic nephritis.

As regards treatment, moreover, the differentiation of the various forms of indurative nephritis does not in the slightest degree help us to a more successful therapy of the disease. The most important thing for the treatment is that the disease should be recognized early, before changes in the circulatory system have begun. The sooner in the disease treatment is instituted the better, for under favorable conditions and early treatment complete involution of the disease may occur, as in the case of acute nephritis, or a remission of the disease with comparative cure may result. When but slight loss of the renal parenchyma has taken place, the healthy parenchyma can compensate entirely for what is lost. We have often seen how, after extirpation of one kidney, the other takes up its function.

I cannot enter into the details of treatment to-day. I shall only say that the main point in the therapeutics of kidney diseases is the diet and the manner of life. Remedies come in only as a secondary matter. It may be set down as a rule that the more recent the case the more absolutely must a thoroughly non-irritating diet for the kidneys be insisted on, and the more firmly must rest of mind and body be required. In older cases the reins need not be drawn so taut. When exacerbations occur, however,—acute renewals of the inflammatory process,—then an absolute diet and rest, by all means in bed, must be prescribed. Where contracted kidney has developed in all its clinical details, patients must be treated in almost exactly the same way as if they had heart lesions.

[Note.—The autopsy of the second case, three days after the clinical lecture, showed a pair of typically large spotted kidneys, with commencing contraction, cicatricial irregularities, and small cysts on the surface.]

COMPRESSED-AIR ILLNESS, CAISSON DISEASE

CLINICAL LECTURE DELIVERED AT THE CLEVELAND GENERAL HOSPITAL, WITH DRAWINGS OF CAISSONS, TUNNEL SHAFT, AND DRIFT.

BY CHARLES J. ALDRICH, M.D.,

Lecturer on Clinical Neurology and Anatomy of the Nervous System in the College of Physicians and Surgeons of Cleveland, Ohio; Visiting Physician and Neurologist to the Cleveland General Hospital and to the Cleveland City Hospital.

GENTLEMEN,—It should not be the aim of a clinical teacher to bring before under-graduates his rare cases. His duty is to show the common, every-day cases of general practice, but occasions arise when a rare and unusual disease may be sufficiently common as to merit consideration at the hands of the clinician. Such an occasion has arisen, and a very rare disease is to be presented to-day for your observation and study. The disease is called "caisson disease," or, more properly, "compressed-air illness." This disease, ordinarily rare, has been quite common in Cleveland during the construction of the new water tunnel beneath the bed of Lake Erie.

Before studying the disorder as manifested in the man before you, it may be well to speak briefly of the history of this nineteenth-century disease. While literature affords some fragmentary observations upon the effect of compressed air on man while going down in diving-bells, yet we find but little that is satisfactory until after the year 1839, when the French engineer M. Triger [1] put to practical use the great principle of compressed air as a safe barrier against water and quicksand. Large bodies of coal were known to exist beneath the valley of the Loire, but above it was a hitherto impassable layer of water and quicksand. The ingenious Frenchman was equal to the task. By the use of compressed air shafts were carried down in safety to the coal below, and one of the greatest

[1] Mémoire sur un appareil à air comprimé. Comptes-rendus, 1864, t. xviii. p. 884.

problems of engineering was solved. The application of the principle to pier-building for bridges was first carried out by Mr. Hughes,[1] an English engineer, in 1850. Compressed air was not used in tunnel-building until 1879. This was in the construction of a tunnel beneath the Hudson River, designed to connect Jersey City with New York City. Soon after this principle was applied to these various engineering undertakings, it was discovered that if the workmen were subjected to too high a pressure or remained too long under pressure even as low as one additional atmosphere, a peculiar, yet tolerably constant set of symptoms developed when they returned to a normal atmospheric pressure. Among scientific men this disorder has received various appellations,—"divers' paralysis," "caisson disease," and "compressed-air illness;" among the workmen, on account of a prominent symptom, "the bends."

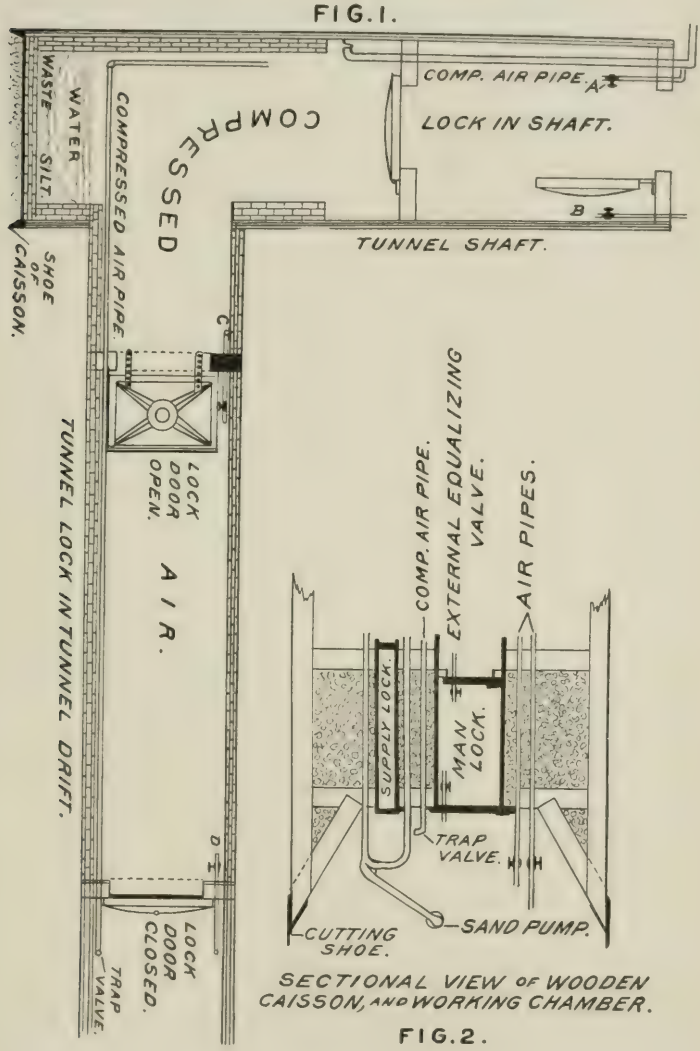
Before passing to a consideration of the disease, I desire to call your attention to these sectional drawings which I have prepared, hoping that they will aid in the explanation of this interesting phase of submarine engineering. Some caissons are made of wood, others of iron or steel. Steel and iron caissons are usually cylindrical. This small figure is a sectional view of a wooden caisson and the working chamber (see Fig. 2). A caisson may be compared to a huge box with the bottom turned upward, and the open space underneath to the working chamber of the former. It is constructed on ways, from which it is launched like a ship, towed to the desired spot, weighted with concrete and masonry until it sinks and its box-like edge, armed with an iron cutting shoe, rests on the bottom of the lake or river. Air-compressing engines are set to forcing air through the compressed-air pipe into the working chamber. Within the chamber this pipe is provided with a trap valve which closes from its own weight and prevents the escape of compressed air in event of any accident to the machinery, thus providing for the safety of the workmen. The air is pumped into the chamber until all of the water has been forced out and the lake's soil lies dry and bare at the bottom of the working-chamber. The chamber is now ready to receive the workmen through manholes left in its structure and which are provided with locks. These locks are supplied with two strong

[1] The Pneumatic Method adopted in Constructing the Foundations of the New Bridge over the Medway at Rochester. Proceedings of the Institution of Civil Engineers, May 13, 1851, vol. x.

doors which swing downward and have air-tight fittings, and each one is pierced by a pipe provided with an air-cock within the lock.

The passing into and out of the working-chamber are called

FIG. 1.



SECTIONAL VIEW OF WOODEN CAISSON, AND WORKING CHAMBER.

FIG. 2.

"locking in" and "locking out;" and as around these transitions is grouped much of the etiology of this disease, I desire you to obtain a correct idea of these procedures. When the caisson is towed

to its position and sunk, the lower door of the lock is closed and the upper one left open, as is shown in the shaft of Fig. 1. Air is then forced into the chamber until the water is displaced and the chamber is ready for the workmen. To go into the compressed-air chamber,—"locking in,"—the men pass through the upper door into the lock, close the door and the air-cock B (Fig. 1), and then open the air-cock A, which allows the compressed air to enter the lock until the density of the air in the lock equals that of the working-chamber below, when the door falls of its own weight and they are free to enter the chamber. In "locking out" the process is reversed. The lock is entered from below, and the lower door and cock A closed. Cock B is then opened to allow the compressed air to escape until the lock pressure equals that of the atmospheric air, when the door falls, allowing the workmen to pass.

When the lake bottom has been well excavated the workmen open one of the air-cocks in the air-pipes, which are seen in the working-chamber of Fig. 2, and allow the air to escape until the great weight on the caisson causes it to settle deeply into the soft mud and silt. In the mean time, and above the water-line, the masonry of the piers is building on the top of the caisson, leaving a passageway for men and material to the working-chamber below. This great weight forces the caisson downward, and is called "settling the caisson." It is aided, however, by the workmen in the compressed-air chamber, who open the valves in the air-pipes (see Fig. 2), allowing the pressure to fall and thus diminish the resistance to the downward course of the caisson. The workmen excavate the silt, which is carried to the surface by means of sand-pumps, blow-pipes, and buckets, until they are again down to a level with the cutting shoe of the caisson, when the air pressure is once more lowered and the caisson allowed to sink. This manœuvre is repeated until a solid foundation is found for the pier. Concrete is then "locked in" and the whole working-chamber is filled; and lastly, the locks are removed and replaced by the same material. When the shaft is sunk for tunnel-building the process varies only in detail.

Did time permit, it would be interesting to recount the early observations of compressed-air illness.

Symptoms.—The symptoms of true compressed-air illness occur only during or after the patient's return to the normal atmospheric pressure. In point of time they may occur during the transition

from the compressed air to the normal atmosphere,—"locking out," —immediately after "locking out;" or they may be delayed several hours. Every grade of caisson disease may be met with, from transient twinges of pain in and about the elbows and knees, to convulsions, coma, and death. The pain is intense, with remissions and exacerbations; is particularly severe in the stomach and about the large joints; the victim is bent double with pain, hence the term, "the bends." The pain is of a peculiar rending character, and is well-nigh intolerable.

The duration of the disease depends largely on the type. The neuralgic cases last from a few minutes to five or six days, although the usual duration is about twelve hours. The duration of the paralytic cases can only be measured by the amount of damage sustained by the central nervous system. Some paralytic cases recover quickly; others perish from exhaustion and cystitis after a lingering, painful illness. Many are permanently crippled. The lethal cases are usually of rapid onset, and death is not often long delayed. Some of the brain types may be transient, and present headache, giddiness, double vision, incoherence of speech, and sometimes unconsciousness and convulsions.

The paralytic forms are atypical, with the greatest tendency to paralysis of one side or both lower limbs. The bladder and bowels are almost certain to be weakened or paralyzed. This often occurs when little or no evidence of paralysis exists elsewhere. Any member of the body may, through paralysis, lose the power both to move and to feel, and yet be the seat of atrocious pain,—a genuine *anæsthesia dolorosa.*

The fatal cases, frequently convulsive from the start, are very severe, and usually develop a deepening coma which ends in death.

Etiology.—The causes of caisson disease may be divided into *predisposing* and *exciting.*

The *predisposing causes* are those which relate to the workman's bodily condition and habits. There seems to be a special predisposition on the part of some persons who are affected by a short exposure to a pressure that ordinarily affects no one, while others appear to enjoy a surprising immunity although exposed to all of the causes recognized as most active in producing this nineteenth-century disease.

It was early remarked that the liability to caisson disease is very

much greater in those engaging for the first time in "pressure work." It is also a matter of record that those who begin with the work when the pressure is low and continue on the work during the sinking of the caisson, and are subject to the consequent increase of pressure, are much less liable to suffer than those engaging when the pressure is high. That this apparent immunity is only relative is shown by the fact that some very serious attacks occur in old hands, and very often without discoverable cause.

Andrew H. Smith,[1] while observing the cases of caisson disease that developed while sinking the piers of the East River Bridge, in New York, was convinced that individuals with a tendency to corpulency were especially predisposed to the disease.

Jaminet[2] insisted, and it is now a common observation of experienced men, that to enter the caisson while fasting is hazardous. Very unpleasant effects are oftentimes felt on entering the caisson with a full stomach, before the lapse of from forty to sixty minutes after the meal.

Douchy[3] and others have insisted that alcohol is a potent cause of the attacks.

Disease or any depression of the vital forces from fatigue, loss of sleep, debauchery, or alcoholic excesses, no doubt increases the liability to an attack.

The *exciting causes* of caisson disease can be said to be those excitations which determine the advent of the attack. There are seven paramount excitants to the disease which we will name in the order of their importance:

1. Degree of atmospheric pressure.
2. Length of sojourn under such pressure.
3. The rapidity of the transition from the condensed to the normal atmosphere,—"locking out."
4. Insufficient lapse of time between leaving the condensed air and returning to the same.
5. Lack of sufficient ventilation of the areas under pressure.
6. Exposure to a damp, chilly air after leaving the lock.

---

[1] The Effects of High Atmospheric Pressure, including the Caisson Disease. New York and Brooklyn Bridge Company, 1873.

[2] Physical Effects of Compressed Air, and of the Causes of Pathological Symptoms produced on Man by Increased Atmospheric Pressure. St. Louis, 1871.

[3] Annales d'Hyg. pub. et de Méd. légale, 1854.

7. Active muscular exertion after "locking out."

It is very doubtful if the disease ever occurs in workmen subjected to less than fourteen pounds pressure, and both its severity and frequency increase in a direct ratio to the rise in the atmospheric pressure multiplied by the length of time exposed to such pressure. Men can usually work with comparative safety for a period of eight hours under a pressure of from fifteen to twenty pounds; under a pressure of from twenty to thirty pounds, six hours, divided into two "shifts" of three hours each; under a pressure of from thirty to forty pounds, two hours, divided into two "shifts" of one hour each; but under a pressure of from forty to forty-nine pounds, two "shifts" of forty minutes each are all that can be borne with any degree of safety. The caissons at Memphis were sunk one hundred and twelve feet below the water level, the deepest caisson work, I believe, that has ever been accomplished, and the pressure reached the highest known to caisson-sinking,—forty-nine pounds. The Eads bridge at St. Louis also demanded a pressure of almost the same amount. One of the shafts of our water tunnel now in course of construction is one hundred and sixteen feet deep, but, because it is in clay, needs little more than forty pounds pressure, enough, being in clay, to furnish many cases of "the bends."

The necessity for a proper lapse of time between the shifts of workmen is very important and should be insisted upon. In the too rapid transition from the condensed to the normal atmosphere —"locking out"—lies a most frequent cause of caisson disease. In fact, it is quite certain that if sufficient time were allowed for "locking out," the accident would never occur unless the exposure to pressure had been prolonged beyond the afore-prescribed limit of time. A time limit should be fixed for "locking out," allowing five minutes for the first twenty pounds pressure, and for each additional pound one-half minute should be added.

Experience has taught "pressure workers" that hot coffee and hot blankets to prevent chill are very useful. At Rob Roy, Arkansas, fourteen men came out of a shaft and laid down on benches in a poorly constructed building, and as the weather was warm went to sleep with little cover; three hours later a "norther," one of Arkansas's cold wind-storms, arose, and nine out of the fourteen men were severely and immediately attacked by "the bends." This is a good illustration of the effects of cold after coming out of the caisson.

Few authorities even mention the importance of active ventilation of the condensed-air area as a preventive of the disease. My attention was called to it by the patient before you, who tells me that whenever experienced workmen strike clay they grimly remark, "It's full of the bends," notwithstanding that they are aware that clay demands less pressure than sand. In the sand at Memphis, although the pressure reached forty-nine pounds, there were fewer cases of "the bends" than in our clay-walled tunnel, with a pressure of about thirty pounds. This was, no doubt, due to the method of excavating by the use of blow-pipes, which demands a rapid change of air in the caisson. It is quite probable also that the loss of air through leaks in the caisson causes a greater amount of air to be forced into the caisson to keep up the pressure, thus perfectly ventilating the narrow place. It is also quite certain that the use of iron cylinders instead of the less tightly made wood caissons gives rise to "bends" more frequently on account of the lessened ventilation. Practical workmen are all aware of this fact.

The first writer who called attention to the importance of ventilation was Hunter,[1] whose thesis concerning the disease as observed during the construction of the Forth bridge, unfortunately, was not published. He is quoted by Snell,[2] whose carefully recorded observations made during the construction of the Blackwall tunnel, London, are of great value.

The following table, taken from Snell's book, *Compressed-Air Illness,* will serve to illustrate the tremendous importance of ventilation, which our tunnel and city engineers have entirely overlooked.

Cubic feet of fresh air per man, per hour, in average daily shift.	Number of days.	Cases of illness.	Estimated cases of illness for one hundred days.
Below 4000	56	16	28.5
From 4000 to 8000	47	9	19.1
From 8000 to 12,000	71	8	11.2
Above 12,000	41	. .	. .

[1] Compressed Air, its Physiological and Pathological Effects. Thesis for M.D. Degree, 1887, University of Edinburgh Library.

[2] Compressed-Air Illness, or So-called Caisson Disease.

Years ago, before electricity was used to light compressed-air chambers, the soot from the lights was one of the terrors of the workmen. The respiratory tract became so saturated that for many months after the expectoration would be tinged with black. It was discovered that this was due to the fact that air under pressure does not circulate. Yet we find that in the lake tunnels the engineers were ignorant of these common facts, or they have reckoned little the value of human life, for the only pipes conveying air to the tunnel ended just beyond the lock, while the workmen were some thousand feet away at the face of the tunnel. And they gravely wonder at the City Hall why there was an explosion in the tunnel and a few lives lost!

When the explosion took place in the tunnel the pipes would admit only nine thousand cubic feet of air per hour to the eight men and two mules. Allowing the mules the consumption of air equal to seven men, we have but six hundred cubic feet of air per man. We have no law in Ohio for the protection of men working in tunnels, but the Humane Society might look after the mules.

On leaving the caisson there is a great increase of heart and lung action; the subject often gasps for breath; he is manifestly ill-fitted for any severe exertion, and abundant experience has demonstrated that many cases have been caused and others aggravated by the error of having the locks low down in the shaft instead of at the top. Triger [1] demonstrated at Chalonnes that when the men made the ascent of seventy feet under pressure to the lock at the top of the shaft, it was done more easily than in the open air. Elevators are now used in deep shafts, and certainly their use is attended by a lessened number of attacks.

Morbid Anatomy.—Intense congestion of the brain and spinal cord is the one constant post-mortem finding in fatal cases of caisson disease. In the cord the distribution is usually very uneven. Spots of localized softening appear and effusions beneath the arachnoid take place. The liver, spleen, kidney, and vessels of the stomach and intestines are engorged with blood. Cavities in the brain and spinal cord have been found which contained fluid consisting of blood serum and white corpuscles, with a significant absence of red

[1] Mémoire sur un appareil à air comprimé. Comptes-rendus, 1864, t. xviii. p. 884.

cells. Small hemorrhages in the nerve tissues have been found also.

Pathology.—The pathology of the affection has caused much discussion. Indeed, it is doubtful if the pathology is always the same. In 1860, Francois [1] suggested that the symptoms might be produced by the entrance of air into the blood-vessels of the brain and spinal cord. Hoppe-Seyler,[2] in 1887, repeated Bert's [3] observation; and "Leyden [4] found (in a case of characteristic hemiplegia) small irregular fissures in the mid-dorsal region, chiefly within the posterior and hinder parts of the latter column. The fissures were filled with round cells, but contained no red blood-corpuscles, and from their well-defined edges it was evident that they were certainly not produced by the proliferation of the cells found within them. The only explanation that is satisfactory or in harmony with their features is, that they were produced by the sudden escape of gas, and were afterwards occupied with the round cell" (Gowers).

This theory of Francois and Bert has strong support in the physical behavior of gases under pressure:

1. That under pressure a fluid like the blood will dissolve an excess of atmospheric gases.

2. That sudden relief of the pressure will cause these gases to separate from the blood and act as emboli to the vessels in which they are confined.

3. In delicate structures like the brain and spinal cord they are very liable to split up and rend the nerve tissue, and hence produce irritant temporary or lasting effects according to the part injured and the extent of the injury.

Andrew Smith [5] has evolved quite an ingenious theory as to the

[1] Des effets de l'air comprimé sur les ouvriers travaillant dans les caissons servant de base aux piles du pont du Grand Rhin. Annales d'Hyg. pub. et de Méd. légale, Paris, 1860, 2d Ser., t. xiv. pp. 279–319.

[2] Ueber den Einfluss welchen der Wechsel des Luftdrucks auf das Blut ausübt. Arch. f. Anat. physiol. u. Wissensch.

[3] Une communication relative à la composition de l'air confiné dans lequel meurent des animaux, quand cet air est comprimé à plusieurs atmosphères. Comptes-rendus Soc. de Biol., 1871. Paris, 1873, 5th Ser., t. iii. p. 66.

[4] Ueber die durch plötsliche Verminderung des Barometerdrucks enstehende Rückenmarksaffection, Arch. f. Psych., 1878, ix. pp. 316–329. Tafel.

[5] The Effects of High Atmospheric Pressure, including the Caisson Disease. New York and Brooklyn Bridge Company, 1873.

disease, but it is not, as yet, fully substantiated by pathologic findings. He believes that the great pressure on the circulation of the outside of the body results in a tremendous engorgement of the vessels of the inner portions of the body with a consequent paralysis of the contractile power of those vessels whenever the pressure is removed. Hence the vessels are left without power to return to their normal diameters after the disappearance of the abnormal volume of blood, and the current slows to stagnation. The lower cord, because of its blood supply, is especially predisposed to this stagnation, with consequent serous effusion into the cord and meninges. The effusion may be hemorrhagic. Many of the pathological facts admit of explanation, and are more in harmony with Smith's theory than the gaseous theory.

Diagnosis.—It is certain that epilepsy, apoplexy, uræmia, alcoholism, and diseases of the heart may be mistaken for "bends," and *vice versa*. Each case demands careful observation and all of the resources of the skilled diagnostician to solve the problem.

Prognosis.—The disease is very dangerous to life and the future usefulness of the victim. Many lives are lost in every one of these great undertakings, many more than contractors are willing to admit, and many men are paralyzed for life. Light attacks are suffered by nearly all the workmen, and it is only when the brain and spinal cord are the seat of the lesions that we get the many grades of paralysis or sudden death. Inasmuch as inflammatory changes are not liable to follow the accident, the resulting paralysis is more remedial than that following other forms of injury to the nervous structures.

Treatment.—The preventive treatment should consist in the employment of sound men to begin the work, so as to become accustomed to the daily increase of pressure, and in sufficient numbers to supply the frequent "shifts" necessary when the pressure becomes high; to regulate carefully the sojourn of the workmen under pressure; to give ample time for "locking out;" to enforce stringent rules as regards eating, drinking, and sleeping; to provide elevators in deep shafts and active ventilation in caisson, shaft, and drift; to prepare deep hot-water baths, hot, stimulating drinks and food, and warm, comfortable quarters for the "shifts" of men as they come from the lock. Foley [1] declared, as the result of his observations,

[1] Du travail dans l'air comprimé; étude médicale hygiénique et biologique fait au pont d'Argenteuil. Paris, 1863.

that the return of the sufferer to the caisson is "a true specific." Some authorities recommend the use of a cabinet, so connected with the pressure machinery that a pressure can be obtained therein equal to that of the caisson, and so constructed as to admit of a very slow and even reduction of pressure. Patients placed in these cabinets immediately lose their pain, vertigo, and other disagreeable symptoms.

Many drugs have been used, but the only ones that have been found of general utility are the anodynes.

Now that we have dwelt upon the disease at sufficient length to enable you to appreciate better an individual example, I wish to read the history of this man, who has kindly consented to come in to allow us to see the manifestations of this peculiar disorder, and to study its phenomena.

J. G., married, white, male, born in Ohio thirty-seven years ago. Family and personal history good. Moderate user of alcohol and tobacco. He has followed "pressure work" as an occupation for twelve years, and has had many attacks of "the bends," but was never before incapacitated for work. His ears were "blown out" several years ago. This is a term used by these men to indicate rupture of the drum. It occurred while he was suffering from a cold and his Eustachian tubes were closed. Each drum presents a pinhole opening and his hearing is slightly dulled. He regards this accident to his ears as good fortune, since he need no longer be troubled to inflate his ears while "locking in," nor to keep up an active swallowing while "locking out," as are necessary with people with intact ear-drums.

On the 22d of last August he travelled by rail all night, and while fasting went into the tunnel early on the morning of the 23d under an atmospheric pressure of forty pounds plus fourteen and seven-tenths pounds of the normal atmosphere, and remained two and one-half hours. Two hours later, after a light breakfast, he returned to the tunnel and remained three and one-half hours. He "locked out" safely, but was attacked by "the bends." He was put into a deep hot-water bath, but vomited, and soon went into convulsions and became unconscious. He was wrapped in hot blankets, and had such other care as they were able to give him out on the crib in the lake.

He was brought to the Cleveland General Hospital on the after-

noon of August 24, 1898. He was perfectly conscious, covered with cold sweat, vomited frequently, and groaned with severe pain. His temperature was 99° F.; pulse, 98. The thoracic and abdominal organs presented no sign of gross lesion. The fourteen ounces of urine drawn by catheter contained a trace of albumen, but the microscope revealed no evidence of renal disease.

Motor System.—He had much pain in the lower limbs, which

FIG. 3.

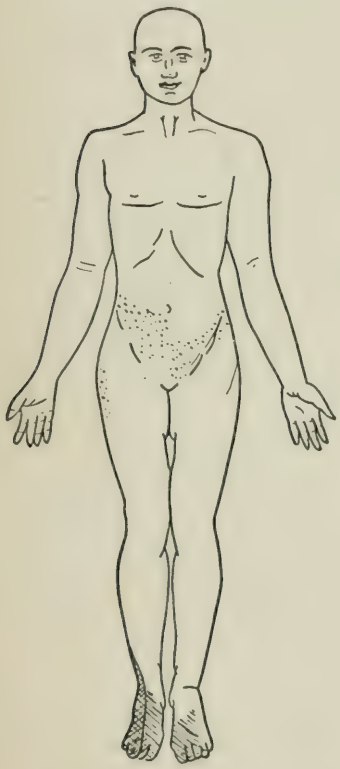
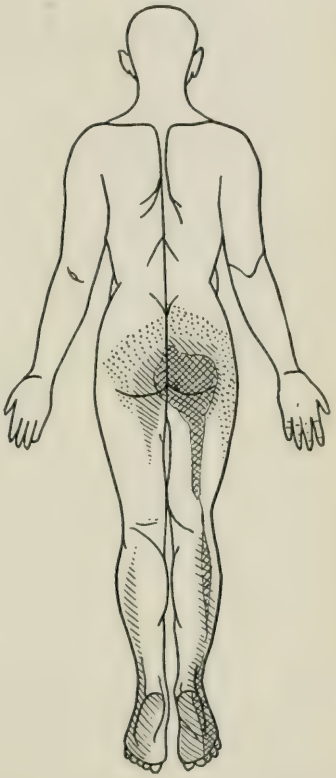


FIG. 4.



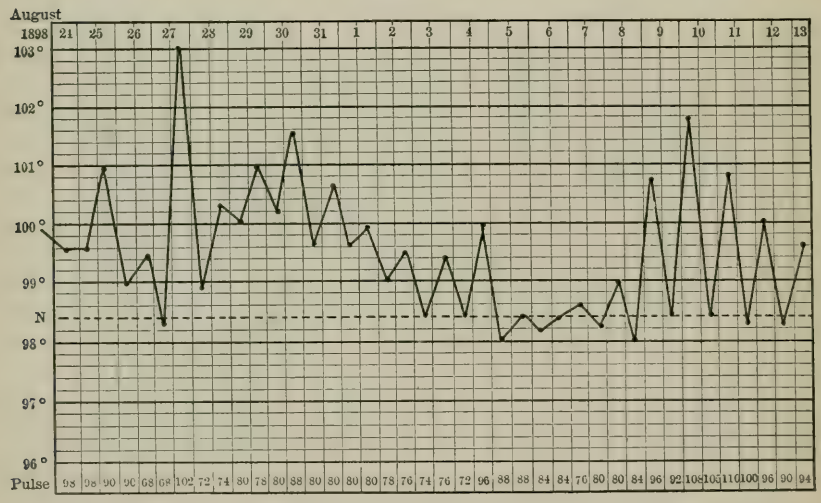
were slightly rigid. He could flex and extend the left leg, but movements of the foot and toes on that side were limited. The muscles of the right thigh, leg, and foot, and also those of the right forearm and hand were powerless. The pectorals and deltoid of the same side were weakened, but not paralyzed. The right arm and forearm, while weak, were not paretic. The neck muscles were unaffected. He had retention of urine, and the anal sphincters were paralyzed

and flaccid. The muscles of respiration as well as the accessories were affected to the extent of embarrassing his breathing. There were no cranial nerve palsies, his sight was unimpaired, and the ocular fundi were normal.

Reflexes.—Skin reflexes of the trunk and limbs, anal reflex, and deep tendon reflexes were all absent.

Sensory Symptoms.—Pains were constant in point of time, but not in location. They were the characteristic pains which I have observed in this disease, and are certainly due to irritation of the posterior roots or root zones of the spinal cord. They came and

FIG. 5.



Temperature and pulse chart of Dr. Aldrich's case of caisson disease.

went with lightning-like quickness, leaving in areas not anæsthetic the same peculiar sensitiveness to touch and pressure that follows like pains in locomotor ataxia. The pains were at times cutting, at others rending, as if the flesh were being stripped from the bones. They were near to but not in the large joints.

He had some loss of sensation in the lower segments of the body which are best shown in Figs. 3 and 4. The crossed lines indicate complete loss of sensation to pain, touch, and temperature; the single lines indicate slight loss of pain and tactile sense; the dotted areas, hyperæsthesia. In the areas of sensory loss, especially in the

right foot and ankle, he suffered much pain. During the next three days he suffered considerable pain, especially in the paralyzed members, but it gradually wore away.

I pass for your inspection a temperature chart of the first twenty days of his illness. The marked rise on August 27 (Fig. 5) is probably due to the occurrence of cystitis. The temperature before

FIG. 6.

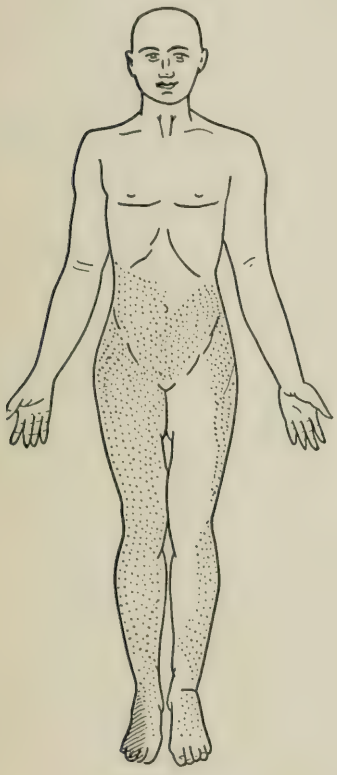
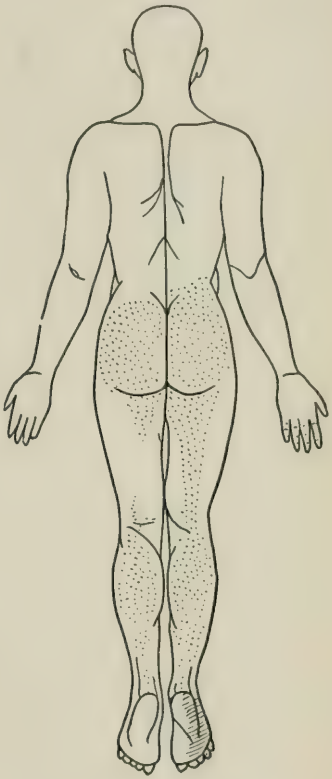


FIG. 7.



that time was doubtless that of the disease *per se*. I will read from my notes of the case:

September 1.—Strength in his left leg has much improved, and he can move the toes of his right foot a little. His cystitis is severe but improving. He has a bad cough and pneumonia is feared. Urine and stools are passed involuntarily. Girdle sensation is quite pronounced, and is irregularly placed about the first and second dorsal vertebræ. Areas of sensory loss unchanged.

October 1.—Both knee-jerks are + and ankle-clonus is present. He can now move foot and toes on the right side. Cystitis is better, and he can hold the urine at times for an hour or two. Constipation is still marked and the anal sphincter flaccid. Girdle sensation now very distinct, and occupies the dotted area shown in Figs. 3 and 4.

November 1.—The bladder and anal functions are better. Power is returning to his legs, and the right arm is nearly as strong as its fellow. Sensory changes are about the same except an increasing hyperæsthesia in both lower limbs, most pronounced in the right leg.

Present Condition.—He is able to walk a little, and will leave the hospital to-day. He has fair control over his bowels, but cannot depend on his urinary sphincter. I ask him to walk, and you see his gait is ataxic. From weakness of his anterior tibial and peroneal groups he fails to lift his toes from the floor, and as a consequence presents a typical drop-foot walk. He still has some little loss of sensation on the outside of the sole of his right foot and ankle, indicated by single lines in Figs. 6 and 7. He has marked hyperæsthesia over the areas marked by dots in Figs. 6 and 7. As I lightly stroke these areas he visibly flinches, yet when I use firm pressure it hurts him less. Knee-jerks + and ankle-clonus is present on both sides. All of the leg muscles are weak, those on the right side, however, most marked. Sexual functions are in abeyance.

His progress towards recovery has been quite satisfactory, and notwithstanding the severity of his attack we are warranted in prognosticating a recovery of all of his lost power and functions.

Note.—Six months after the delivery of the above lecture I learned that our favorable prognosis was justified, as he was then at work and experienced but little inconvenience from the very serious attack.

THE INADEQUACY OF THE PHYSICAL SIGNS AS INDICATING THE GRAVITY OF PNEUMONIA.

BY ANDREW H. SMITH, M.D.,
Of New York.

In a disease having such distinctive physical signs as we encounter in a typical case of pneumonia it is natural to rely in great measure upon these signs in forming our estimate of the gravity of a particular case. Or rather, I should say, we are prone to accept the extent of the consolidation as indicated by the physical signs as an index of the severity of the attack. The area of percussion dulness and of bronchial breathing, unconsciously to ourselves perhaps, is in a great degree the measure of the peril to which we feel that our patient is exposed. We watch the increase of this area with grave anxiety, and note its diminution with intense satisfaction. And in general we are right enough in this. *Other things being equal,* the danger is pretty fairly apportioned to the amount of lung involved.

The actual degree of this involvement, however, is not always easily determined. It happens to all of us with more frequency than is quite comfortable that an autopsy brings unexpected revelations as to the condition of the lungs. In many persons the respiratory murmur in health is very feeble, sometimes almost inaudible even with forced respiration. In such persons the initial crepitus of pneumonia may easily escape detection, and the later bronchial breathing is not well pronounced. This is especially true when there is a thick layer of adipose tissue overlying the chest. In the latter case the percussion dulness also is obscured. But even under the most favorable circumstances and with the best trained ear it must be admitted that the resources of auscultation and percussion scarcely supply the data for a fine and accurate appreciation of what is going on in the lungs. The methods at best are but clumsy ones. The affected area may be at a distance from the surface of

the lung and separated from it by a considerable thickness of tissue yielding sounds of varying significance.

The resiliency of the chest wall is a variable factor at different ages and under different conditions. The presence of pulmonary emphysema or of old pleuritic adhesions may complicate the results of auscultation and percussion.

Add to this the restriction which consideration for the comfort and perhaps the safety of the patient may impose upon a careful and thorough physical examination, and it will readily be seen that valuable as the physical signs undoubtedly are, they are by themselves quite inadequate to a full exposition of the changes taking place in the lung. But aside from the difficulties just mentioned there are points regarding the physical signs which I believe to be very important. If we could in every case determine the limits within which consolidation had taken place, we should still be uncertain as to the full import of the condition present. For the area of consolidation may, and often does, fall far short of the area in which toxine is being formed. So long as the disease is advancing in the lung, there will always be a zone outside of the consolidated area in which the amount of exudate in the air-cells will not be sufficient to cause notable signs, but in which, nevertheless, an active formation of toxine is progressing. The slightest film of fibrin upon the wall of the vesicle will harbor a colony of pneumococci whose toxine will be under the very best possible conditions for absorption. Unlike the fully filled alveoli, these vesicles will have an active circulation going on in their functional capillaries, and the toxine as it is formed will be immediately taken into the circulation. Nor is this all. The younger the organisms the more virulent is the poison formed by them, so that while cultures taken from the centre of a consolidated area show little toxicity, those taken from the circumference of such a patch present the maximum of virulence.

Now as the toxæmia in pneumonia plays quite as important a part as the restriction of the breathing-surface, it is clear that, so long, at least, as the local process is spreading, there may be serious mischief going on which will give very little if any indication of its presence through the medium of the physical signs. Auscultation over this advancing zone will afford only occasional moist râles which would scarcely attract attention. More significant might be, per-

haps, a diminution or suppression of the vesicular murmur, a sign not much dwelt upon, but which sometimes precedes the crepitant râle. Percussion would be of little avail, as the dulness would shade off imperceptibly from the hepatized area. But whatever may be the actual condition of the lung itself, the question as to the gravity of a pneumonia includes many other factors. The most important of these is the soundness of the patient before the attack. Pre-existing morbid conditions probably determine the outcome of a very large proportion of fatal cases. Thus of seven cases occurring in subjects of chronic kidney disease in the Presbyterian Hospital, only one recovered. Of eleven cases with pre-existing valvular hyperplasia eight proved fatal. Five patients with cirrhosis of the liver all died. Four cases in phthisical subjects were fatal. In these examples the result evidently depended in large measure upon conditions that bore no relation to the physical signs of the pneumonia.

The lesson we are to learn from this, it appears to me, is that while the physical signs in pneumonia are very valuable in establishing the diagnosis, they cannot be relied upon altogether to indicate how sick the patient really is. On this point the information to be derived from the rational symptoms is quite as important. This is especially true in the earliest stage. Even before the chill, or before there is any considerable rise of temperature, there is in from twenty-five to thirty per cent. of the cases a prodromic stage marked by malaise, headache, anorexia, dull pains in limbs and back, and perhaps epistaxis and diarrhœa. This stage may last for two or three days or more, during which time there will be absolutely no change discernible by auscultation or percussion. The only thing to point to the lungs may be a slightly increased ratio of the respiration to the pulse. If we see the patient during this stage, which we seldom do, abortive treatment may be eminently successful. But if we wait until the physical signs make the diagnosis absolutely certain the golden opportunity may be lost. And here I would emphasize the unwisdom of repeated and exhausting physical examinations after once the diagnosis is established. The progress of the disease from that time on can readily be followed by the symptoms with very little aid from the signs; and thereby much distress and, it may be, no little danger can be avoided. Especially should the consultant, if called by a competent practitioner,

forego the opportunity for a display of thoroughness, and, accepting for the most part the statement given to him of the physical signs, base his advice upon the symptoms and the general aspect of the case.

What needs most carefully to be looked for is the occurrence of pulmonary œdema or of pleural or pericardial effusion, and having these possibilities always in mind we do not need a frequent resort to physical examinations to assure ourselves in regard to their presence or absence.

As to the consultant, by the time his services are required the important question will be, not how much or how little pneumonia is present, but how the patient is bearing it, and this will be answered more by the symptoms than the signs.

A CASE OF MEDIASTINAL TUMOR, ILLUSTRATING DIFFICULTIES OF DIAGNOSIS.

CLINICAL LECTURE DELIVERED AT THE COLLEGE OF PHYSICIANS AND SURGEONS, CHICAGO.

BY ROBERT H. BABCOCK, A.M., M.D.,

Professor of Clinical Medicine and Diseases of the Chest in the College of Physicians and Surgeons, Chicago, Illinois.

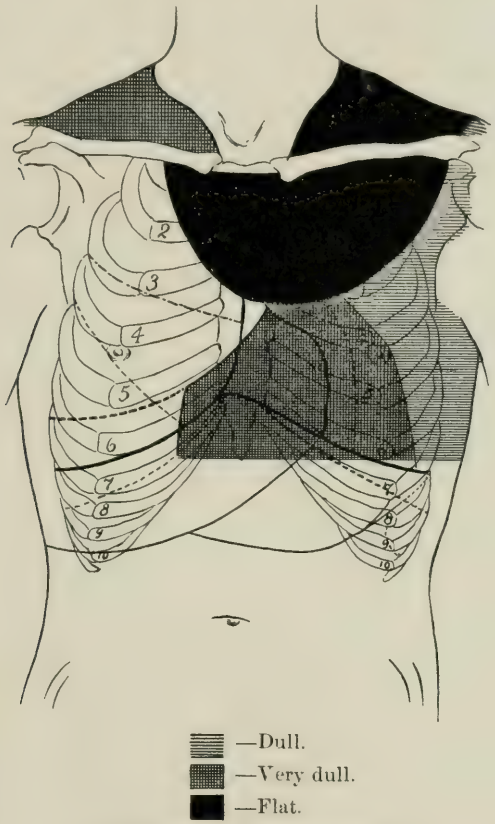
GENTLEMEN,—We have this morning a case presenting the symptoms and physical signs of intrathoracic pressure. The patient is a male, forty-seven years of age, German, hotel porter, unmarried; family history unimportant. His illness dates from last Christmas, or thereabouts, previous to which time he had never been seriously ill, and he denies ever having contracted gonorrhœa or syphilis. During the holidays he had a cough, which he attributes to la grippe. The cough subsided, and his health remained good until the middle of April, when he discovered one morning that he was hoarse, and this hoarseness has continued until the present time. Early in May he began to suffer from pain in the front of the chest, chiefly in the left infraclavicular region. The pain gradually increased, and became so severe by the middle of June that he sought admission to the Cook County Hospital. After one week's rest the pain abated and the patient asked for his discharge. On July 4, 1899, he resumed his work, but was obliged to quit at the end of a week on account of weakness and increased pain. He has attempted work only once since that time, and with the same result. In the middle of August he was admitted to the Dunning Infirmary, where he has remained until the present time. Soon after his admission the pain became less, and was succeeded by dyspnœa. Cough was not a very troublesome symptom during the weeks he was suffering from pain, but he was never free from it. Soon after the middle of August both the cough and the pain subsided, and did not reappear until October 1. Some pain of a neuralgic character, being worse on cold, damp days, is still present.

During the summer and early fall the patient lost strength, but in the two weeks prior to October 1 he gained seven pounds in weight. His weight on admission to Dunning was one hundred and three pounds, about the middle of September one hundred and two pounds, and on October 3 one hundred and nine pounds. The symptoms of which he complains most are constant dyspnœa, or a subjective sense of insufficient air, and an occasional slight cough, not distinctively paroxysmal, but increased by exertion and the act of swallowing. Expectoration is difficult, the sputum muco-serous. There has never been any hæmoptysis. Dysphagia is present in a slight degree. Liquid food can be swallowed, but some difficulty is experienced with solid food. Pain at the present time is by no means a marked symptom. Weakness is, however, pronounced. The appetite remains fair. There is constipation, but no indigestion. The urine is scanty, but contains neither albumen nor casts. There is no headache; there has never been nose-bleed nor tinnitus aurium. The temperature is normal, ranging from 98° to 99° F. This comprises the history and symptoms.

As you see, the patient presents some well-marked signs of intrathoracic pressure,—congestion of the face, distention of the superficial veins, chiefly those of the neck, the external jugulars; the veins on the anterior surface of the chest are visible rather than palpable, but those of the arms are very turgid. The pupils are equal, normal in size, and responsive to light. Emaciation is not marked, and instead of the sinking of the supraclavicular and infraclavicular spaces seen in phthisical patients, there is prominence above and to the left of the suprasternal notch, and also fulness of the chest over the manubrium both to the right and to the left of the sternum, a little more pronounced at the second left costal cartilage. The bulging begins four centimetres to the right of the sternum, passes downward to the fourth left costal cartilage, and, curving upward, reaches the left clavicle twelve centimetres from the sternum. There is no pronounced distention of the abdomen. Marked pulsation is not visible, but it may be felt in the upper portion of the chest. The pulsation does not seem to be expansile; it is sudden rather than slowly heaving. There is a sense of great resistance, but no thrill.

The radial pulse is small and accelerated, varying from 100 to 110. The left radial is shown by the sphygmograph to be ampler

than the right, but the character of the tracing of the radials is practically normal. The carotids are equal. Distinct tracheal tugging is not present, but inspection and palpation reveal a slight displacement of the trachea towards the right. Careful palpation of the cervical triangles detects a flattened gland about the size of a large pea just outside the left scalenus. One or two of the other glands



Dr. Babcock's case of mediastinal tumor.

are somewhat indurated. On the right side, there is a gland about the size of a small shot. The right post-cervical gland is palpable, and about as large as a No. 6 shot. The axillary glands are not enlarged; on the left side they are just palpable. The right epitrochlear gland is indurated but not enlarged. The inguinal glands are indurated. Examination by percussion and auscultation reveals

rather interesting changes. On the front of the chest the region corresponding to the bulging area already described is absolutely flat. Outside the flat area on the right side there is resonance as far as the base of the lung or the upper border of hepatic dulness at the sixth rib. On the left side, below the area of absolute flatness, there is dulness extending laterally to the anterior axillary line and downward to the base. From the level of the fourth costal cartilage on the left side, the line of dulness passes down and out towards the right, becoming continuous with the upper border of hepatic dulness a little to the right of the sternum. Laterally on the left side the percussion-note is impaired. On the right side it is resonant. Posteriorly on the left side there is marked dulness nearly to the scapula. Below that the note is resonant. On the right side dulness, less pronounced than on the left, extends to the infrascapular fossa, and below the scapula there is the usual pulmonary resonance, a trifle exaggerated perhaps. Examination of the liver by percussion shows that dulness passes down into the epigastrium, a hand's breadth below the ensiform appendix. In either hypochondrium tympany is encountered at the lower costal margin, a little outside of the mammillary line. The area of splenic dulness does not seem to be increased, its upper border being in the usual location.

Upon auscultation over the flat area in front the heart's sounds are heard with difficulty at its centre; they are more distinct at its edge, particularly on the left. The second sound is accentuated. There are no murmurs. In the neck also the heart sounds are feeble, and there are no murmurs. Over the precordia the heart sounds are very indistinct, but they become clearer towards the epigastrium. In the median line a blowing murmur may be heard, and the systolic impulse is recognizable. Auscultation of the lungs in the right suprascapular region reveals bronchial breathing, but no râles. Over the anterior portion of the chest on the left side the respiratory sounds are heard with difficulty; over the area of flatness they are entirely absent, but they become audible again along the outer axillary portion of the chest. Posteriorly, there is bronchial breathing at the apex. Below the area of dulness, where the lung is resonant, the respiratory murmur is exaggerated and fine crackling râles are heard; these are most distinct around the lower angle of the scapula, over the middle portion of the lower lobe; they have a moist

quality, very suggestive of the fine râles of pulmonary œdema. Respiration is accelerated, averaging from 30 to 36; it is somewhat labored and is accompanied by some inspiratory sinking, especially at the lower portion of the chest.

Examination of the larynx reveals paralysis of the left vocal cord, congestion of the larynx, and slight œdema of the epiglottis.

The symptoms and physical signs, therefore, point to pressure within the thorax, and the area of flatness indicates that its probable cause is to be sought within the anterior mediastinum. There can be little doubt of the existence of a body in the anterior mediastinum causing pressure upon all the neighboring viscera. The left lung is evidently pushed upward and backward, and is œdematous. The right upper lobe also shows evidence of pressure, but it is not so marked as in the left lobe. The shape of the chest and the distention of the thoracic veins indicate obstruction to the return circulation. The paralysis of the left vocal cord from pressure on the recurrent laryngeal nerve of that side, the slight dyspnœa, particularly on exertion, and the deviation of the trachea from the median line to the right point to mechanical obstruction. The area of cardiac dulness and the location of the heart sounds indicate a displacement of the heart downward, with its base apparently a little to the right, so that the heart lies not only lower, but more horizontally than it should, and probably occupies the space between the diaphragm and the anterior chest wall.

We know that pressure may be due to aneurism of some portion of the thoracic aorta, to new growths (carcinoma, sarcoma, lymphosarcoma, and osteosarcoma), to mediastinal abscess, and to some rare conditions, such as dermoid cyst, lipoma, fibroma, and enlargement of the mediastinal glands, as in Hodgkin's disease. Aortic aneurism, cancer, sarcoma, and mediastinal abscess are the diseases most frequently encountered in this locality. As to the malignant tumors, there is some difference of opinion as to the relative frequency of cancer and sarcoma. Hare's review of five hundred and twenty cases of mediastinal tumor apparently indicates a preponderance of cancer; but these statistics show that the diagnosis in many of the cases reported was made at a time when sharp distinctions were not drawn between carcinoma and sarcoma, and is consequently open to some doubt. Eichhorst says cancer is more common than sarcoma, while others are of the contrary opinion.

Mediastinal abscess occupies the third place in point of frequency in Hare's statistics. As the other conditions mentioned are so very rare, we will confine ourselves to the differential diagnosis of aortic aneurism, malignant tumors, whether cancerous or sarcomatous, and mediastinal abscess.

All these conditions may produce similar symptoms,—symptoms occasioned by pressure. The pathognomonic symptoms of aneurism may be absent when the sac of the aneurism is occupied by blood-clots, as it then forms a practically solid tumor. When the aneurism is saccular and is not occupied by clots the physical signs of aneurism may be well marked, particularly after the tumor has attained a considerable size. During the early stages of development the symptoms of intrathoracic tumor of whatever nature may be so vague as to lead to a diagnosis of bronchitis, on account of the cough, expectoration, and slight dyspnœa. At times they closely resemble those of the incipient stage of pulmonary tuberculosis. The condition of the lungs need not be taken into account in this case, because the symptoms and physical signs point unmistakably to a growth of some sort in the anterior mediastinum. The symptoms of aneurism depend upon the direction of the growth of the sac,—that is, whether the aneurism develops centrifugally or centripetally. If it develops from the centre towards the anterior parietes, we have bulging and the signs of pressure on the chest wall. If it involves the ascending aorta, the prominence appears to the right of the sternum in the so-called aortic area. If, however, it arises from the transverse aorta, the direction in which pressure is exerted is from the median line to the left of the sternum, in the second left interspace. The tumor caused by aneurism of the descending portion of the aorta occupies a position to the left of the vertebral column at about the level of the eighth dorsal spine.

If the direction in which pressure is exerted be inward and there be nothing specially distinctive revealed by examination of the lungs, the heart must be carefully auscultated. Certain adventitious murmurs, or bruits, and modifications of the normal heart sounds, are generally present in aneurism. There may be a systolic murmur and, when the aortic valves are incompetent, a diastolic murmur.

Both heart sounds, but most frequently the second, may be intensified, or murmurs and sounds may be variously combined. If

the tumor press forward, it may be accessible to palpation. The character of its pulsation is then often significant, being expansile, —that is, the pulsation seems to cause the mass to expand in all directions, not in one only. There may also be a thrill.

These pressure-effects upon the heart, lungs, and adjacent viscera differ in no way from those produced by solid tumors of malignant character in the same situation. In solid tumors, however, there is no thrill, and, though pulsation is sometimes perceptible, it is never expansile. In this case the heart sounds are feeble, and, with the exception of accentuation of the second sound, present nothing anomalous. There are no signs or symptoms peculiar to aortic aneurism. All the symptoms present might be due to a solid tumor as well as to an aneurism. When the transverse arch is implicated, pulsation is marked in the suprasternal notch and tracheal tugging is a very significant sign, although it is not pathognomonic of aneurism in that location, for it sometimes accompanies aortic insufficiency, and has been observed in mitral disease. When present it indicates pressure by the aneurism on the trachea and left main bronchus. If the sac be so filled with clots that there is not a wide excursive movement, tugging may be absent, and in this instance tracheal tugging cannot be positively determined. In the case of malignant or solid tumors within the mediastinum the heart sounds are not distinct over the area occupied by the tumor, as they are in aneurism. They may, however, be feebly propagated through the tumor mass. Pulsation may be present, but it is not expansile. A bruit may be produced by the pressure of the solid tumor on the aorta or other large vessel. Tumors, whether due to aneurism or to cancer, may press on either bronchus, according to the direction in which the tumor develops, or upon the trachea, lungs, or one or both of the recurrent laryngeal nerves. In the present case the tumor has developed forward, forcing the borders of the lung aside, and we have an area of flatness, especially marked in the region of the manubrium and to the left of it. There is nothing in the situation of this area that is peculiar to either class of tumors, but its uniform semicircular outline suggests aneurism rather than a malignant growth, as the latter usually causes irregularity in the outline of the area of dulness. It is also stated that solid tumors usually produce greater turgidity of the veins, especially the superficial ones. This is a general declaration which surely does not hold

good in all cases, because an aortic aneurism filled with clots is practically a solid tumor, and the direction in which this tumor develops will determine the extent and degree of the pressure-symptoms. If it causes constriction of the superior vena cava or the innominate or azygos vein, just as pronounced distention of the veins of the chest and abdomen is produced as from a solid tumor.

Although authors make rather sharply defined distinctions in physical signs, identical symptoms and physical signs may be produced by both kinds of growth. Mediastinal abscess may, however, be excluded by absence of fever.

There are certain facts in the history, symptoms, and physical signs of the case before us which point in one direction as much as in the other, and I wish to dwell on these for a few moments, to see if with their aid the diagnosis can be established.

There is no history of syphilis, which is perhaps of no special importance; but as syphilis is supposed to predispose to aneurism, it must not be ignored. On the other hand, the man has been a hotel porter, and the strain incident to his calling might well lead to aortic aneurism. The first symptom noted by the patient was aphonia, which developed about the middle of April, since which time hoarseness has been continuous, and the left vocal cord is paralyzed. It is possible that this paralysis was the cause of the aphonia. This would point to an aneurism of the transverse arch. Then the effects of pressure manifested themselves on the anterior chest wall, making pain a prominent feature; after the middle of August the pain ceased, the cough grew less, and dyspnœa became more marked.

Now this seems to indicate that pressure was first exerted upon the anterior chest wall, and that then, for some reason, its direction was changed towards the centre. Dulness over the upper and resonance over the lower lobe show not so much occlusion of the main bronchus as simple collapse of the upper lobe from pressure. Cough, at first due to pressure upon the trachea and perhaps upon the lung also, gradually ceased, because compression of the lower part of the trachea became for a time less marked. Cough is probably due in this, as in most cases, to bronchial or tracheal irritation, rather than to irritation of the lung itself.

This sequence of symptoms seems to indicate that the mass causing pressure underwent an alteration in form and size that

caused a change in the direction of the pressure it exerted. This also points to aneurism. Furthermore, the patient has not been troubled by expectoration, has had no hæmoptysis, and no rise of temperature; all of which speaks for aneurism, because malignant tumors, especially cancer of the encephaloid variety, are prone to involve the lung, and cause expectoration sometimes distinctly bloody, sometimes containing cancer cells. Disintegration and ulceration of the lung are also more likely to occur in cancerous affections than in aneurism. Yet cases of aortic aneurism which have exerted sufficient pressure on the lung to cause softening and even gangrene are not uncommon. Malignant tumors often produce pleurisy or pericarditis with effusion, with consequent febrile temperature depending in degree upon the severity of the infection and upon the changes going on in the lungs. The absence of such conditions and symptoms in this case also points to aortic aneurism.

Furthermore, the amplitude of the left radial pulse, as compared with the right, possesses a certain degree of value, since in aneurism of the arch inequality in the pulse is not uncommon, in consequence of partial occlusion of one of the large branches, either by a blood-clot or by a twist in the vessel. On the other hand, slight enlargement of the cervical glands on the left side, and the absence of bruit and of expansile pulsation, favor a solid tumor. Solid tumors, moreover, are very likely to produce great asthenia and emaciation, patients becoming cachectic. Aortic aneurism may also lead to emaciation and weakness, but they are not so pronounced as when due to malignant tumors. In this case, although the patient has lost strength, he has distinctly gained in weight during the last two weeks, which would exclude malignant tumor and favor aneurism. So you see, we are confronted by conflicting conditions; and yet when we weigh the evidence for and against, and when we consider the possibility of a sac changing its form and direction of pressure in consequence of the formation of clots in the sac,—when we take all this into consideration, the preponderance of evidence seems to be in favor of aortic aneurism, although I would not commit myself absolutely to this diagnosis. It may occur to you to ask, Why do you not determine the differential diagnosis by means of the X-ray? And it is a pertinent question. The X-ray is of service, and if the patient's strength admits it will be employed. The X-ray shadow demonstrates merely the presence of a foreign body;

it does not indicate its nature. Fluoroscopic examination might show some pulsation of the mass; if it did, it would settle the diagnosis.

[One week after the presentation of this patient before the class he died suddenly. The necropsy was made thirty-one hours after death. Epitomized, the post-mortem findings were as follows. The body was that of a fairly well-nourished man. There was no bulging of the chest wall, and the superficial veins had collapsed. The subcutaneous tissues of the chest were somewhat œdematous. Occupying the anterior mediastinum was a mass composed partly of creamy yellow puriform fluid and partly of enlarged, soft, fleshy glands. Between the apex of the left lung and the trachea there was a cavity filled with purulent fluid and having roughened, shaggy walls. The left upper lobe was infiltrated with irregular, moderately soft, yellowish masses which had almost entirely replaced the normal pulmonary tissue, while the pleura was firmly adherent. The lower lobe was œdematous and congested, the pleura was adherent to the diaphragm, and the pleural cavity held about a pint of limpid fluid. The right upper lobe was firmly indurated and the overlying pleura was puckered and adherent. There were old adhesions at the base, but the rest of the lung presented nothing anomalous.

In the pericardium there were a few enlarged, soft yellowish glands. The aorta was apparently healthy, and the heart, displaced downward, was soft, but to the naked eye healthy. There was nothing of importance in the abdominal cavity. This tumor then was not an aneurism, but a lymphosarcoma which had probably become infected.]

PLEURISY WITH EFFUSION, AND ITS TREATMENT.

CLINICAL LECTURE DELIVERED AT RUSH MEDICAL COLLEGE, CHICAGO.

BY JOHN A. ROBISON, A.M., M.D.,

Assistant Professor of Medicine at the Rush Medical College, President of the Chicago Society of Internal Medicine; Attending Physician to the Presbyterian Hospital, etc.

GENTLEMEN,—The case which I present to you to-day is interesting on account of the presence of certain physical signs which might be misleading. The patient is twenty-two years of age; the family history is negative so far as any hereditary element is concerned; her general health, previous to the present illness, has been good; her habits of life are regular. On November 13 she was taken ill; her indisposition was preceded by a period of malaise characterized by headache, pain in the limbs, loss of appetite, sluggishness of the bowels, and a slight cough. Eighteen days ago she had a chill, unaccompanied by pain. At the time of the patient's admission to the hospital she was well nourished; the skin was warm, the countenance flushed, the pulse rapid and regular, the respiration regular, the tongue coated, the digestion and appetite good. She complained of extreme thirst, but had no pain or distress in breathing. The physical examination revealed the following conditions:

Enlargement of the left side of the chest, with diminished movement and effacement of the intercostal spaces; displacement of the heart to the right, the apex-beat being about an inch to the right of the ensiform cartilage.

On palpation vocal fremitus was absent over the lower portion of the left lung, and greatly diminished over the upper portion.

On percussion there was flatness anteriorly, extending from the costal arch to the clavicle. Posteriorly there was flatness over the lower portion of the chest, with dulness towards the spine and the upper part of the suprascapular region.

On auscultation there was puerile respiration on the right side,

with absence of the respiratory murmur and voice-sounds on the left side below the third rib. There was broncho-vesicular respiration above the third rib,—rather more bronchial than vesicular,—and the voice-sounds were slightly increased in intensity.

The pulse was 120, rather weak and thready, but regular; temperature, 100.4° F.; respirations, 24. The next morning at eight o'clock the pulse was 96; temperature, 100.8°; respirations, 22. Since admission to the hospital the pulse-rate has risen from 100 to 112, the temperature from 100.2° to 100.3°, the respirations from 28 to 36. The patient has at no time complained of shortness of breath or of pain.

The diagnosis is doubtless evident to you: the case is one of pleurisy with effusion. I wish to call your attention to two facts in the history of the case,—the absence of pain and of orthopnœa, or shortness of breath. The patient maintains that she has not suffered, and that she feels first rate. Therefore, two prominent symptoms which are present in nearly all cases of pleurisy with effusion are absent in this case.

Pain is one of the first symptoms noticed by patients suffering from pleurisy. It may, however, be slight in degree when the first stage, that of hyperæmia, is of short duration or is rapidly followed by effusion of serum into the pleural cavity, which prevents friction between the inflamed surfaces of the costal and parietal pleuræ, and thereby eliminates this source of pain. The absence of orthopnœa in this case may be due to the fact that the effusion came on early and developed gradually, giving the patient's thoracic organs time to accommodate themselves to the changed condition. The compression of the lung and displacement of the heart have therefore given rise to no inconvenience from intrathoracic pressure.

About twelve days after the inception of the illness three pints of clear serum were aspirated. The patient was placed upon quinine sulphate, two grains three times a day, and one-tenth of a grain of calomel every half-hour, with an occasional saline cathartic. Four days later it was thought advisable to aspirate a second time, as the apex of the heart had again moved to the right of the ensiform cartilage, and the physical signs demonstrated the effusion of more fluid. The operation was therefore repeated and twelve hundred cubic centimetres of clear serum were removed, and the patient has been feeling comfortable ever since.

We now come to the second interesting feature in the history of this case. This (illustrating) is the point of the apex-beat before aspiration. This (illustrating) is the point of the apex-beat at the present time, about an inch to the left of the median line. On percussion we obtain practically normal resonance over the right lung. At the apex of the left lung we get dulness, accompanied by a peculiar resonance, called Skodaic resonance, which is due to incomplete consolidation of the lung by pressure and diminution of the vibrating area. I believe the tympanic quality of the percussion note is caused by recession of the relaxed lung from the chest wall. Dulness is not nearly so pronounced now as it was a few days ago, when it was very marked. Percussing posteriorly below the third rib, the region over the scapula is extremely dull; below the scapula it is flat.

From the physical signs present one should expect to find entire absence of respiratory murmur and voice-sounds, at least as high as the spine of the scapula. The converse, however, is true, and it is the second interesting feature in this case. There is absence of the respiratory sounds from below upward as far as the fifth rib, where bronchial breathing begins. This extends almost to the apex of the lung. The respiratory murmur is blowing in character and higher in pitch than normal; the expiratory sound is prolonged. There is also bronchophony. This, under ordinary conditions, would certainly suggest the presence of consolidation, probably inflammatory, of the lower lobe of the lung. That, however, cannot be the case here, for there is no past history nor present evidence of pneumonic inflammation. Pneumonia begins with a severe chill, which is followed in a few hours by a high temperature lasting usually for several days, and in the majority of cases falling rapidly between the fifth and eighth days. The fever disappears sometimes by crisis and sometimes by lysis. Inflammation of the lung is also accompanied by blood-stained sputum and cough. The absence of these symptoms enables us to exclude pneumonic inflammation. This form of resonance is not infrequently present in pleurisy with effusion, and is due to the fact that the lung is practically consolidated, not by an inflammatory process, but by pressure; just as a sponge filled with water, and then squeezed, is solid. By nearly freeing it from air and water a mass results that is solid and compact, but spongy in consistence.

This case is, therefore, interesting because it presents the physical signs of consolidation, but no symptoms of inflammation. This has an important bearing upon the treatment. If the pleurisy was accompanied by inflammation of the lung, the case would be much more grave. The treatment of pleurisy with effusion is simple, except when the inflammation of the pleura is due to tubercular disease, in which event the problem which confronts us is far more serious and the prognosis less favorable.

In order to establish a rational theory of treatment, it is necessary to consider, first, the etiology of the disease; secondly, the pathological conditions which are present; thirdly, the physiological action of the remedies; and fourthly, the part which Nature plays in restoring the patient to health.

The etiology of the disease determines the treatment. If the case is one of simple pleurisy, pleurisy *a frigore,* or subacute inflammatory pleurisy, then the treatment is comparatively simple. If the inflammation is due to the pneumococcus or other pathological germs, the treatment indicated in the simple form is somewhat modified; if to tubercular infection, the treatment is altogether different.

In the first, or hyperæmic, stage of the disease, the capillary vessels are filled with blood; the pleural membrane is reddened and somewhat thickened; secretion has ceased and the surfaces are dry. The nutrition of the epithelial cells is interfered with, and they are swollen and clouded.

These pathological conditions give rise to certain definite signs and symptoms, the first being usually, as in this case, a slight chill. The indication, therefore, is that the patient be put to bed and kept warm and comfortable. A cotton jacket should be provided and poultices applied. A very convenient method of maintaining a uniform degree of heat over the chest is by the use of a Leiter's coil through which water of the desired temperature is caused to circulate. A piece of flannel or cotton batting should be placed between the coil and the patient's chest. The continuous heat causes dilatation of the capillaries of the skin, and thus lessens the hyperæmia of those of the pleura.

Secondly, the pathological conditions present during the first stage in the majority of cases cause pain. That symptom was absent in this case. The pain is most quickly relieved by morphine

or codeine, given hypodermically or by the mouth. The old-fashioned Dover's powder, or the syrup of Dover's powder, is also an excellent remedy. Opium not only relieves the pain, but lessens the irritation at the point where the inflammation exists; it reduces the hyperæmia, increases the activity of the secretion of the kidneys, and promotes general diaphoresis.

The third symptom is rapid breathing, which is an almost invariable accompaniment of the first stage of this disease, and which causes increased intrathoracic friction. To lessen this, the chest should be strapped. This procedure restricts the respiratory movements and places the inflamed tissues at rest.

Saline cathartics should be given to cause determination of the blood from the inflamed pulmonary surface to the intestinal tract. Mercurial preparations are also often of benefit.

The second stage is characterized by an effusion into the pleural cavity. This causes a rapid separation of the pleural surfaces, the pain and dyspnœa disappear, the patient feels relieved, and is apparently getting well. So long as the patient is comfortable refrain from interfering; but when the intrathoracic pressure becomes so great as to interfere with the action of the heart or of the respiratory organs, what is to be done? In simple inflammatory pleurisy it is not, as a rule, advisable to remove the fluid until *positive* symptoms of pressure are present. The majority of cases of this type run a uniform course, the fluid ceases to develop, and the pressure reaches its maximum after from fourteen to sixteen days; the fever and fluid then disappear. If, however, after the lapse of several days or weeks there are positive indications of intrathoracic pressure, the fluid should be removed by aspiration. What are the signs of positive intrathoracic pressure? They are, first, effacement of the intercostal spaces; secondly, bulging of the affected side and flatness above the third costal cartilage. Both of these signs are present in this case. Other signs and symptoms are displacement of the heart and of the diaphragm downward; extreme shortness of breath; disturbance of the circulation, evidenced by carotid and jugular pulsation; interference with the return circulation of the blood, shown by cyanosis, blueness of the lips and finger-nails, cold extremities, and a short, jerky cough, accompanied by tenderness and pain and a tendency to syncope. When these signs are present something must be done at once to relieve the pressure or the pa-

tient's condition will become very grave. The danger is always greater when the effusion is on the left side, on account of the displacement of the heart to the right, and interference with its free action. Another source of danger is the liability of the opposite lung to œdema.

When these signs exist you should aspirate. The use of trocar needles obviates all danger of wounding the internal organs. Never aspirate while the patient is in the sitting posture, as the danger of collapse and syncope is great.

The point of election is a matter of small moment; it is generally determined by the condition of the patient and the character of the disease. The most accessible place, as a rule, is in the sixth or seventh intercostal space, behind the posterior axillary line on the left side, and a little higher in the same relative position on the right side. Sometimes the effusion extends as far down as the eleventh space, when the needle may be introduced at a point below the angle of the scapula. It is not absolutely necessary to remove all the fluid, as in many instances the removal of a very small quantity suffices to relieve the pressure symptoms, and to enable the absorbent vessels to carry on their work with greater activity. In this case we removed three pints of fluid at the first operation. The fluid rapidly reaccumulated, necessitating another aspiration a few days afterwards. The physical signs to-day do not indicate reaccumulation, so that the patient will not be aspirated again.

Another point to remember is that the patient should be placed upon a diet which tends to lessen the accumulation of fluid. Few patients will submit to the dry diet treatment of pleurisy. Skimmed milk is a natural diuretic, and given with a minimum amount of water acts very satisfactorily. Another remedy which in these cases almost always secures positive results is the syrup of the iodide of iron. The iodide of potassium is recommended by many; the same alterative action and a good chalybeate effect in addition are obtained from the iodide of iron. From thirty to forty minims should be given three times a day. The patient should be kept in bed until the fever and the effusion have disappeared, when he may resume his ordinary habits of life.

PARACENTESIS THORACIS FOR PLEURISY WITH EFFUSION; TRICUSPID REGURGITATION.

CLINICAL LECTURE DELIVERED AT THE NEW YORK POLYCLINIC HOSPITAL AND MEDICAL SCHOOL FOR GRADUATES.

BY WILLIAM H. KATZENBACH, M.D.,

Professor of Diseases of the Chest and Internal Medicine at the Polyclinic; Vice-President of the New York Academy of Medicine.

Gentlemen,—We have here to-day the patient whom I showed you at the last clinic, and who, you will remember, we decided, was suffering from pleurisy with effusion. There has been no improvement in his condition, but, on the contrary, his symptoms have grown worse, the difficulty of breathing being a little more marked and the heart farther displaced towards the right. As there is now some bulging in the intercostal spaces and dulness extends to the upper border of the second rib anteriorly and to the spine of the scapula behind, we shall evacuate the liquid from his pleural cavity to-day.

We have in this case some of the typical indications for tapping the pleural cavity. The effusion has lasted some time, or at least we must believe so from the history. Marked dislocation of the heart has occurred, nearly the whole of one pleural cavity is filled with fluid, and there is considerable difficulty in breathing after even very slight exertion. Ordinarily in acute pleurisy it is well to wait two or three weeks before removing the fluid. Even then its evacuation should not be undertaken if absorption is going on, unless symptoms have developed that seem to demand it, or fever continues as a persistent complication.

We have had in this case a not infrequent, though not the usual, mode of development of the pleurisy and of the consequent effusion. Commonly, pleurisy begins with an acute pain accompanied by fever. Sometimes the initial symptom is a distinct

chill. This chill is not, however, so severe as that which occurs in pneumonia and the fever is not so high, though it lasts longer. The sputum of pleurisy, when any is expectorated, is mucoid in character and very different from the rusty sputum of pneumonia.

In this case we have the other mode of development exemplified. The beginning was insidious, and no special symptom occurred to mark the inception of the pathological process in the pleura. The patient, who is about thirteen years of age, says that he has felt uncomfortable for some weeks, has had no appetite, has been restless, has become tired on slight exertion, has not slept well at night, has had difficulty in breathing on extra exertion, as climbing stairs and the like, and has had a sensation of fulness in the left chest. He has no cough, no chill, no pain.

Pleurisy attacks individuals of all ages. It may occur in children less than a year old. It has been known to occur in people of eighty or ninety. It is most common between the twentieth and fortieth year. The reason for this would seem to be the greater exposure of patients at this age to the causes which usually bring on a pleurisy. Of these, the most important is exposure to cold. It has often been noted that pleurisy develops on the side of the chest which has been exposed to a draught or which has undergone rapid abstraction of heat. While we generally believe that pleurisy is microbic in origin, some such predisposing cause as exposure to cold seems to be essential to afford the bacteria an opportunity for growth.

The signs of the pleurisy in this case are very distinct. The patient is thin, and the interspaces between the ribs, while easily traceable, are much less noticeable on the left side than on the right. There is bulging of some interspaces and diminished respiratory movement. On percussion we get a markedly dull, high-pitched note, besides the sense of resistance to the finger that is such a significant physical sign in a case of pleurisy. The elasticity that should be imparted to the tissues by the presence of air in the lung immediately beneath the finger which acts as plexímeter is absent. The dull note and the sense of resistance can be traced to the upper edge of the second rib in front. The apex-beat of the heart can be felt here to the right of the sternum, about two inches from the right nipple, and there is evidently considerable interference with its action from the presence of fluid

in the left chest. Vocal fremitus and vocal resonance are also absent.

The normal breath-sounds are entirely masked over the dull area, and we hear only bronchial breathing and bronchophony, so that we evidently have to do with a considerable amount of fluid in the pleural cavity compressing the lung.

For paracentesis, instead of any complicated apparatus, I prefer to employ an ordinary Davidson syringe which is attached to a Flint trocar and canula, the latter being provided with a stop-cock. A glass tube is inserted in the rubber tubing in order to detect the flow of liquid. This serves all ordinary purposes. I have used it in a very large number of cases, and have no reason to believe that complicated apparatus is more efficient in securing evacuation of the pleural cavity. I have the area of the chest wall into which I propose to plunge the trocar thoroughly disinfected. Then, about an inch and a half from the tip of an ordinary Flint trocar of large size I place a ring of adhesive plaster, in order to guard against penetrating into the chest farther than necessary. After inserting the trocar and canula I remove the trocar, turn the stop-cock, and by compressing the bulb fluid is withdrawn from the chest. Should the canula become stopped by flakes of fibrin, I reinsert the trocar, so as to secure an open passage through the canula.

A few minutes previous to the thoracentesis the patient is given a hypodermic injection of morphine and atropine, principally to prevent cough, but also as a prophylactic against shock. He is then placed in a recumbent position with the affected side lowermost and at the edge of the bed or table. Two or three pillows are used to support the head and shoulders, and the hand of the unaffected side is drawn forward so as to rest on the opposite shoulder. It is best to make the introduction of the canula as painless as possible, and this is accomplished by anæsthetizing the site of puncture with a spray of ether or ethyl chloride. As the skin is somewhat hard to penetrate, a preliminary incision is made through it with a scalpel. This is best done by pinching up a fold of the skin, inserting the scalpel, and then drawing it outward.

The incision should be made at the upper part of the intercostal space selected (in this case the eighth), so that the skin may

be drawn down before the insertion of the canula through the underlying tissues, thus making a valvular opening which may be readily sealed after the operation. The trocar and canula are thrust with a quick movement into the pleural cavity. Withdrawal of the trocar is followed immediately by a flow of serum. The stop-cock is then turned and the tube of the Davidson syringe screwed to the canula. The bulb of the syringe is then compressed, with the production when released of a vacuum. This starts the flow of liquid, and as the distal end of the syringe is considerably lower than the patient the siphonage force which is exerted makes the stream continuous. The discharge of fluid may be regulated by the stop-cock.

The fluid may be withdrawn as rapidly as is desired, but more can be obtained by a slow, small stream. If a more or less continuous stream is kept up, that is sufficient. Should the patient show signs of discomfort or should difficulty of breathing, or coughing develop, the stream may be stopped for a moment by turning the stop-cock on the canula. If after resuming the withdrawal of fluid coughing recurs, the canula should be withdrawn.

The principle on which this apparatus works is practically that of the siphon. The syringe bulb is used to start the flow, and later may be of service in assisting the flow when it becomes sluggish, or if for any reason it should stop; but this rarely happens. The valves as arranged for the ordinary syringe prevent regurgitation of fluid and also the ingress of air. This piece of glass tubing inserted in the rubber tubing on the proximal side of the bulb helps us to gauge the rapidity of the flow and to detect any stoppage as soon as it takes place. Usually, as in this case, there is no necessity to use the bulb for pumping.

The relief of pressure upon the heart and upon the great blood-vessels in the thorax by the removal of the fluid may cause disturbance of cerebral circulation, and even lead to collapse. To guard against this accident half an ounce to an ounce of whiskey should be at hand, as a stimulant. The patient should be reassured from time to time in order to prevent as far as possible all excitement that might affect his heart action and all irregularity of breathing. The head should be kept low and the operation stopped if syncope occur.

The fluid has a greenish tinge and is turbid, and is not as thin as we should expect to find it in so young a subject. Perhaps the fact that it is some time since the effusion took place has something to do with this alteration in appearance. There is, however, no evidence of pus. You will find this slightly greenish tint not infrequently; though the fluid usually found in a case of recent effusion is of deep straw color, clear, and rather thin.

The amount of fluid that should be withdrawn in any given case will depend entirely on the amount present. With this apparatus I have taken away eighty ounces of a serous effusion out of the pleural cavity, and in a case of sero-purulent effusion I once removed one hundred and fifty-three ounces. The general rule is to continue the removal of the fluid until it ceases to come away freely, or until cough or sense of constriction in the chest appears, when the canula should be removed and the wound of entrance covered with a piece of iodoform gauze. The object of paracentesis thoracis is not to remove all of the fluid present, but to take away sufficient to relieve the symptoms caused by the presence of the fluid, to allow the heart to come back to its normal position in the thorax, and to permit the lung to expand once more. Usually when the pressure upon the lymphatics in the pleura has been removed they proceed promptly to take up whatever fluid may be left in the pleural cavity until all of it has disappeared.

After the removal of the canula the skin is allowed to return to its normal position, closing up the wound in the deeper tissues, and then the cutaneous wound is sealed with collodion and a pledget of cotton fastened over it with a strip of adhesive plaster. After this we shall put the patient to bed, and require him to remain there for the next two or three days.

Already we can notice, since the fluid has been removed, that the apex beat has changed its position considerably to the left. For the first few hours we shall insist that he shall lie absolutely quiet and not move for any reason. There is very little danger in the operation of paracentesis of the thorax, and the reported deaths (which are very rare) would seem to be due to collapse incident to the sudden change in pressure relations within the thorax interfering with the cerebral circulation, or to the occurrence of cardiac or pulmonary thrombus.

After two or three days we shall begin to take measures to

further secure the normal expansion of the lung. For this purpose respiratory gymnastics are very important, and the patient should be taught to take long breaths slowly and to retain the air as long as possible. I have found such directions very useful in those cases of pleurisy in which the expansion of the lung is hindered by the presence of fluid or by adhesions of pleural surfaces. It must be borne in mind, after all, that normal inspiration exerts very little, if any, pressure upon the lung. Whatever pressure there is, is neutralized by the elasticity of the pulmonary tissues, and so fails to produce expansion. In forced expiration, however, the pressure of air in the normal lung passes through the tracheal bifurcation into the other, the collapsed lung, and so aids mechanically in distending it.

Where patients are delicate and can afford to do so, I often recommend after thoracentesis that they shall go to some high altitude. At an altitude the rarefaction of the atmosphere leads to greater expansion of the lungs and so aids in overcoming the collapsed state.

After the fluid has been removed tonics are needed and should be given freely. I have found the combination of quinine, iron, and strychnine very useful in these cases, and recommend it frequently. Our patient here will be directed to take it for some time.

The prognosis in a case like this is reasonably good. The remaining fluid and the fibrous exudate are in time absorbed. The lung will resume its normal position and function, and may probably be no more susceptible to disease than the other lung. The one element in the case that obscures an otherwise favorable prognosis is the uncertainty of the etiology of the disease. There was a time when we considered that uncomplicated serous pleurisy was a simple inflammatory condition, and not necessarily due to the invasion of microbes. We now know that this is very rarely the case,—not even in cases of traumatic pleurisy. There is always an infective agent at work. According to circumstances, it is the tubercle bacillus, the pneumococcus, the streptococcus, or some other pathogenic bacillus.

It had been noted that following an attack of pleurisy patients not infrequently developed tuberculosis. Then it became apparent that the pleurisy itself was primarily tubercular or very frequently

secondary to a tuberculous process in the lung. It is well to search carefully for physical signs of the presence of tubercles in the apices of the lungs. Not infrequently careful investigation will disclose the presence of a previously unsuspected tuberculosis in this region.

Later on, after the discovery of the tubercle bacillus, it became customary to examine the fluid removed from the pleural cavity, in order to demonstrate the presence or absence of tubercle bacilli. These were found occasionally, but not as often as might have been expected from the clinical claims that had been made of a connection between pleurisy and tuberculosis. It was found, however, that even when no tubercle bacilli could be detected under the microscope, and none could be demonstrated even by the most careful culture methods, if some of the pleuritic fluid were injected into susceptible animals they developed tuberculosis in considerably more than half the cases. It seems advisable then to warn patients who have had pleurisy of the possibility of the development of tuberculosis even when no physical signs of disease are present. They should be warned not to overexert themselves, to spend a good deal of time in the open air, to live as far as possible only in well-aired houses and rooms, to eat heartily, and on the occurrence of the first sign of loss of weight or any constitutional disturbance, as fever or loss of appetite, to seek medical aid. From what we know of the climatic and open-air treatment of consumption in our day, it will be comparatively easy to save such patients from the development of the disease to any serious extent, if they follow, with a reasonable amount of care, the directions given them.

For some time after the patient gets up there will be certain abnormalities in heart action that it is well to warn patients of beforehand, in order to avoid too great solicitude on their part with regard to symptoms that may develop. During its period of displacement the nervous supply of the heart has been somewhat interfered with, and it has acquired certain habits of palpitation and irregularity that are liable to recur on exertion or excitement. The tachycardia which develops on even slight exertion is a notable feature of certain of these cases, and calls for the use of heart tonics.

[At the next clinic the patient was exhibited, and showed marked improvement. The measurement of the two sides was equal, the heart apex was on the left of the sternum, the left chest

expanded more. Dulness was present to the level of the site of puncture, and flatness below. Vocal fremitus was also absent. Bronchial respiration was replaced by feeble distant vesicular murmur. There was still a slight amount of fluid present in the chest.

The patient will be kept in bed for a day or two longer. Then he will be allowed to get up and go around, in the hope that the increased activity of metabolism and of circulation, lymphatic as well as hæmic, will favor absorption of the fluid.

If after some days of respiratory gymnastics the collapsed lung does not resume its normal position completely, the other side may be strapped for some time with adhesive plaster, in order to decrease the respiratory excursions on the right, and so increase the amount of movement and respiration on the left. In a young patient like this, however, it is very doubtful if any such bothersome method of treatment will have to be adopted. It is well to bear this expedient in mind, however, as it will prove of good service in delicate adults whose pulmonary tissue lacks normal resiliency.]

Our next patient to-day is a man of forty, who, until nine months ago, followed his occupation of pressman on a newspaper. This involved the lifting of large rolls of paper not only heavy in themselves but awkward to handle, and so required him to make strong muscular efforts at short intervals. His work was severe rather than laborious. About nine months ago, as stated, he developed shortness of breath, especially on severe or unusual exertion, as going up stairs and the like, and when required to lift particularly heavy weights. General weakness also began to develop, most noticeably in his legs, and because of this he finally had to give up his occupation.

On inspection two things are particularly noticeable: first, there is a pulsation in the neck; and, secondly, there is a pulsation in the hepatic region on the right. As to the pulsation in the neck, we are at once tempted to think of aortic regurgitation, and we look for the heaving impulse in the cardiac region which so frequently accompanies this condition. While the apex beat is displaced downwards and to the left, as it would be if aortic regurgitation really existed, and is more diffuse than normal, it can by no means be said to be a heaving impulse. When we examine

the pulse, we find it rather weak and by no means full. There is no sign at all of the *pulsus celer,* the Corrigan water-hammer pulse, that we should expect to find if aortic regurgitation existed.

Percussion shows increased cardiac dulness which extends from the apex beat slightly beyond the nipple-line in the seventh interspace to almost an inch beyond the right border of the sternum. There is evidently a considerably dilated right heart. The positive and relative heart dulness is also increased upwards to the left of the sternum, so that it extends to the upper border of the third rib. There seems then to be also a dilated left auricle.

On auscultation we find that the second sound of the heart is completely drowned by murmurs. The systolic murmur can be heard best near the apex beat, and is conducted around to the axilla and even to the angle of the scapula. The murmur is more distant and not so distinctly heard at the aortic cartilage. A softer systolic murmur can be heard also over the ensiform cartilage, and it is conveyed for some distance up the sternum. This taken in connection with the pulsation of the liver would seem to indicate that regurgitation is taking place at the tricuspid orifice. No murmurs are conducted up into the vessels of the neck.

When we examine the pulsation in the neck, we are at once struck with the fact that it occurs in vessels that are much more superficial than the artery. The external jugular veins are seen to be widely distended, and in them it is that the pulsation occurs. How different this is from pulsation in the arteries can be seen by comparison with this other case, in which aortic regurgitation undoubtedly exists and in which there is marked carotid pulsation. When I shut off the flow of blood by pressure on the vein here just above the clavicle, so as to stop the reflux of blood from the innominate vein, the pulsation stops at once. If I exert pressure higher up, the pulsation becomes more marked. This is really the diagnostic sign of the presence of a leakage at the tricuspid orifice, but it occurs only when the valve at the orifice of the jugular vein as well as the tricuspid has become incompetent.

The heart murmurs in this case are very interesting. That of the mitral is heard most distinctly at the apex, and is carried around into the axilla and can be heard at the angle of the scapula. It is rough and blowing in character. The tricuspid murmur is much softer in quality, somewhat musical, and is heard best at the

lower end of the sternum, a little to the right, and then can be followed upward towards the right shoulder. The difference in the quality of the sounds of the two murmurs is a good diagnostic sign. When taken in connection with the way in which the two murmurs are conducted, it is pathognomonic of the presence of the corresponding lesions.

As a rule, heart murmurs alone are not of such great diagnostic importance as they were once thought to be. It is the general condition of the heart that must finally decide the prognosis in valvular lesions. Here, however, in this case we have dilatation of the heart, which points to the fact that a pathological process is at work in the muscular mechanism. When besides this we are able to distinguish between different murmurs that exist at different orifices, we have substantial data for diagnosis that cannot lead us astray.

The condition of the liver in this case is extremely interesting. It is not only pulsating, but it is tender and noticeably enlarged. Usually the lower edge of the liver reaches to about the costal border in the parasternal line. Here, by gentle percussion we can demonstrate that its lower border is at least two inches below the lower edge of the costal cartilages, and it is distinctly palpable. The enlargement of the liver is due to the damming of blood in it. This causes a venous engorgement of the organ, and the irritation thus set up leads eventually to the formation of new connective tissue in the midst of the liver substance. The picture presented post mortem in such cases is the so-called nutmeg liver—the yellowish-brown mottled liver due to the deposit of hæmoglobin in the liver-cells as a consequence of the stagnation of blood in the hepatic vascular system for long periods of time. There is another condition for which this might be mistaken, if the heart lesion failed to be recognized and if a clear history of the case were not obtained. It might be diagnosed hypertrophic cirrhosis if there was a history of alcoholism in the case, and if this and the enlargement of the liver were alone taken into account. In this case, however, there can be absolutely no doubt as to the diagnosis.

We have then in this case a series of signs which point undoubtedly to the existence of mitral regurgitation and tricuspid regurgitation. Mitral regurgitation is the commonest of all the acquired heart lesions. Tricuspid regurgitation except when con-

genital is always secondary to mitral regurgitation. It does not indicate, as a rule, an actual lesion of the tricuspid valves, but rather a dilatation of the orifice, so that the valves cease to be functionally capable of closing it completely even when fully distended for that purpose. This failure of function leads to the backing up of blood in the venous system, and this is usually first noticeable in a pulsation of the liver. A pulsation of the veins of the neck, as in this case, usually follows if the tricuspid regurgitation continues for some time to any serious extent, and the valve at the root of the jugular is also incompetent.

Besides these valvular troubles there is another interesting picture to which I wish to call your attention. If you examine the apex beat carefully, instead of noting the usual impulse, which slightly raises the chest-wall and then allows it to fall back, you will observe that there is at each beat of the heart a retraction of the overlying tissue. This retraction is quite noticeable when attention has been called to it. Its significance is not always easy to determine. Pathologically it usually means that there is a synechia pericardia,—that is, an adhesion of the parietal layer of the pericardium with the visceral or heart layer of the membrane, and the consequent obliteration of the pericardial sac. This nearly always leads to dilatation of the heart.

In the treatment of this case a number of things must be taken into consideration. In the first place, we do not advise treatment merely because we find a heart murmur and are able to demonstrate the existence of a valvular lesion. These conditions are never sufficient of themselves to indicate direct treatment. There must be present some symptom of the effect upon the general health of the patient, of the interference with the general circulation, owing to the defect in the valvular mechanism of the heart. In the case before us, we have, of course, a series of such symptoms. They are not unlike the symptoms that are usually set down as characteristic of failure of cardiac compensation.

Some time ago,—nearly a year,—the patient had to give up work because of increasing weakness, noticed most in his legs. There were also shortness of breath, a tendency to persistent cough, slight hæmoptysis, and swelling of the feet. The pulmonary symptoms were due to backing up of blood in the lungs through the mitral orifice. The tendency to dropsy was due to the re-

gurgitation of blood into the general venous circulation by the failure of the tricuspid valves.

One of the most important indications in a case of this kind is to relieve the venous engorgement of the liver. This can be best done by one dose, or a series of doses, of calomel, given at short intervals. The use of calomel in heart cases generally where compensation is failing is one of the important heritages from our medical ancestors that has been too much neglected. The relief afforded the portal circulation by the effect of the calomel in the intestines reacts marvellously upon the general circulation and makes the work of the laboring heart much easier.

There is another indication, and a very important one, for calomel in these cases. One of the things especially to be borne in mind in all cases of heart trouble with failing compensation is the effect that disturbance of the circulation has upon the kidneys and upon the elimination of the toxic products of metabolism in the urine. In all heart cases the urine must be carefully watched. As soon as it becomes lessened in quantity the indication is to give diuretics, to increase the amount of liquid that the patient is taking, and to direct that he should live in an equable temperature, in order to encourage perspiration. Calomel seems to have a special effect in these cases in producing an increased amount of urine, though, of course, where the amount of urine is distinctly lower than normal other diuretics should also be used.

As to the heart itself in this case, there are certain indications for treatment that should not be neglected. The heart is pulsating between 110 and 120, and, besides, it is irregular in its action. Our patient has been taking digitalis for some time; and while he stands the drug well, it has ceased somewhat to have its physiological effect upon the heart. In such cases it is often advisable to change to another of the heart tonics, such as strophanthus. I have found, however, in a good many cases that strophanthus and digitalis given together act better than either one of these drugs, and when combined with strychnine all three formed the best heart tonic that we have. We shall give our patient the following prescription then:

℞ Tr. digitalis,
Tr. strophanthus,
Tr. nuc. vomicæ, āā ʒi.
Sig.—Twenty drops three times a day, increasing.

Besides the medical treatment, however, a most important part of our duty in all cases in which heart lesions exist is to give patients explicit directions as to their mode of life. Where compensation has once suffered they must be strictly forbidden to take tobacco, tea, or coffee. They should avoid violent exercise,—to run to catch a car, to ascend stairs hastily, and the like. They must be taught that the simplest and most nutritive diet, containing a certain amount of liquid, and with as little stimulants as possible, is best for them if they wish to remain in good health. In a word, their lives are crippled, and they must learn to live so as to make as few calls as possible upon the impaired vitality which remains to them if they hope to live in reasonable comfort. The lesson seems an easy one to give, but you will find it very hard to impress it sufficiently upon your patients to make them appreciate at times in their after-life, when cardiac compensation is so perfect as to make them feel as though nothing were the matter with them.

SOME OF THE SOURCES OF ERROR AND DANGER IN THE USE OF THE ASPIRATOR.

BY J. N. HALL, M.D.,

Professor of Medicine in Gross Medical College; Visiting Physician to the Arapahoe County Hospital and to St. Anthony's Hospital, Denver, Colorado.

The disappointment of failing to find fluid with the aspirator when it has been confidently anticipated is so keen that I wish to mention some of the sources of avoidable error. Even with the greatest care the result may be negative when fluid is actually present; and we should minimize the chance of this occurrence to the utmost.

So long as a good vacuum can be maintained, it is immaterial what particular form of aspirator is employed. Whatever the apparatus used, it must, of course, be proved to be in good working order before the operation. I have seen a slight leak in a joint utterly defeat the purpose of an otherwise well-devised aspiration. Leakage is especially likely to occur at the valves and at points where rubber tubes are used to join together the glass or metal parts. Both the sharp needle, of one to two millimetres diameter, and the canula and sharp trocar, so arranged that air may be excluded, are recommended by equally good authorities. The advantages of the former are greater ease of introduction and greater simplicity in operation. But, if clogged, the hollow needle must be withdrawn, cleared, and reintroduced, while the canula may be cleared *in situ;* there is also more likelihood of wounding the expanding lung with the sharp needle as the fluid is withdrawn, and thereby at times exciting violent cough, necessitating the withdrawal of the needle.

I know of a case in a prominent hospital in which air under pressure was injected, without apparent harm, fortunately, into the pleura, owing to a mistake made in the application of the tube to the pump. To avoid such a possibility, and to make certain of a good vacuum in the reservoir, always test the aspirating tube in

a basin of sterilized water or salt solution beforehand. The apparatus is more certain to work well with the tube thus filled, since it minimizes the danger of leakage, and the liquid is probably less dangerous than air if drawn into the pleural cavity.

The canula may become clogged in several ways. A bit of floating fibrin may be sucked into the open end, and some effusions are very difficult to deal with because of such obstruction. A purulent effusion may be so thick as to pass with great difficulty through the tube. The fluid occasionally coagulates so quickly that a solid clot forms in the canula during the time that the pump is being used to renew the vacuum, and the flow is consequently interrupted. In these cases the stop-cock must be closed and the blunt needle introduced into the "head" of the canula, the cock on the proximal side of the lateral opening to which the aspirating tube is attached then opened, the needle pushed through to clear the canula, and, before it is entirely withdrawn, the cock closed again, to prevent possible admission of air to the cavity. As a matter of fact, air admitted in this way is not often a source of serious harm.

As the fluid is withdrawn, the expanding lung often floats down against the orifice of the canula and blocks it. In this event the tube must be partially withdrawn or its direction changed, when the flow commonly begins again. Many attempts at aspiration fail because the canula is not in the free fluid, even though it may have passed through it. In many pleuritic exudates the angle between the chest wall and the liver is more or less filled by a deposit of fibrin. If in such cases the needle be inserted too low, no flow results. Not uncommonly the point traverses free fluid, but strikes the diaphragm, the liver, or the spleen, and, the suction being cut off before the needle is withdrawn, the result is negative. It is best to withdraw the needle slowly in such a case while continuing the aspiration, when frequently the flow begins as the point becomes freed from the tissue in which it has been imbedded.

It is not uncommon for failure to result because the lung is adherent through an old pleurisy, over a small space in the middle of a large area of flatness. In such cases, if the lung be not solidified, a light bloody fluid mixed with air is obtained. Careful percussion beforehand should enable us to avoid this error.

If the cavity be long and narrow, as, for instance, in interlobular pleurisy, its fluid contents, even when amounting to several

ounces, may be entirely missed, if the needle passes to one side or to the other. In old pleurisies the deposit of fibrin may be so thick that a short needle fails to reach the fluid. The inch-long hypodermic needle is too short to be depended upon, and a longer one should be used.

Effusion is sometimes, but rarely, attended by slight negative pressure, so that the fluid will not flow unless vigorous aspiratory force be applied. I think this cause is not operative when the aspirator is used, it being insufficient to prevent the withdrawal of the fluid, although with the siphon it has occasionally caused failure. I have known air to be sucked into a trocar in the attempt to dislodge a piece of fibrin which had obstructed it, the blunt needle having been withdrawn too far before closing the stop-cock, but I have never seen harm from this cause.

I once made an autopsy for the coroner in a case in which a consumptive had died a few hours after an irregular practitioner had introduced a large trocar into the second right interspace, apparently in the attempt to treat a cavity by aspiration. The right pleural cavity was filled with blood, which had come from an intercostal artery which had been nearly severed by the needle. The bleeding had been continuous from the artery because of the inability of its coats to contract and check the hemorrhage. This is the only instance in which I have seen or known serious hemorrhage from such an accident in aspiration. I once saw the entire left chest filled with blood from an intercostal artery which had been severed by a pistol-shot, the woman escaping death only through timely surgical intervention. It is well to avoid even the slight chance of puncturing such a vessel. Fortunately, punctures in solid organs are almost devoid of harmful results if the needle be aseptic. Although I have not, to my knowledge, seen the heart punctured, this accident is reported to have occasionally occurred without serious result. Certainly, pure blood has been withdrawn at times, indicating that, if not the heart, a large vessel has been opened. When the lung is punctured, or, more often, when it is torn, pneumothorax may, and does infrequently, result. The fact that the lung is commonly more or less completely solidified in the cases in which aspiration is done no doubt accounts in part for this immunity from harm. One of the advantages of the trocar over the hollow, sharp-pointed needle is that if the expanding lung should come against it, especially in

the act of coughing, there is much less liability to puncture of the pleura and the lung. On the other hand, pneumothorax seldom follows puncture by the small needle, while it is not rare after puncture by the larger trocar. New membranes may be so vascular as to bleed freely after puncture with the larger instrument, and possibly with the smaller, although the danger is less in the latter case.

It is said that the costal pleura may be pushed in front of the needle instead of being pierced by it, especially if it be introduced with a boring motion. It is certainly not a common accident.

Periostitis may follow injury to the rib by hugging it too closely to avoid the artery under the rib above, but it is not common. When the skin is very thick, so much resistance is sometimes felt when the shoulder of the canula engages as to suggest impact upon bone.

Neither I nor any of the friends with whom I have discussed the subject have met with the albuminous expectoration so much dreaded by the early French writers. It is practically never seen when the fluid is withdrawn slowly and in moderate amount only, since it results from too sudden and too great disturbance of circulation in the lungs long subjected to the pressure of an intrathoracic effusion. With these precautions the dangers of œdema and syncope are also reduced to a minimum, and we rarely encounter serious pain, cough, or constriction of the chest. It is safer to desist whenever any of these symptoms appear and are persistent. I have known of a fatal result from disregard of this rule.

If careful and repeated puncture fails to produce fluid, one must think of solid intrathoracic growths. The needle enters very differently if these be present, since the resistance is much greater, and after its introduction it cannot be as freely moved as in a cavity containing fluid. In such cases other signs will soon be found if the possibility of cancer or sarcoma be borne in mind.

The most puzzling of all cases, I think, are those in which, with more or less solidification of lung, there is a sacculated pleural effusion. Nothing but free use of the needle will determine this.

It is best to turn the stop-cock before withdrawing the needle, because of the greater danger of leaving a fistulous tract if pus be aspirated into the channel left by the needle.

ELEPHANTIASIS ARABUM.

BY C. C. MAPES, M.D.,

AND

FRANK K. GREEN, Ph.G., M.D.,

Of Louisville, Kentucky.

Elephantiasis Arabum is so seldom encountered in this part of the world that it is regarded as a pathological curiosity; therefore, our knowledge of its clinical manifestations, physical characteristics, morbid anatomy, causation, etc., is of necessity based more upon text-book and statistical information than upon actual observation and clinical study. The unreliability of medical statistics renders knowledge so acquired of little practical value, but close study of occasional cases enables a critical and careful observer to separate the wheat from the text-book and other statistical chaff, and thus arrive at fairly accurate conclusions.

Elephantiasis is essentially a chronic, circumscribed hypertrophy involving the skin and subcutaneous tissues, often resulting in gigantic enlargement of the affected parts. As an endemic disease, it occurs only in tropical climates; sporadic cases are occasionally encountered in all parts of the world. From published reports it appears that there are no characteristic pathological or clinical differences between the endemic and the sporadic types. When incised, the affected areas in either variety are found indurated, the density being uniform throughout the superficial and deeper structures so far as the hypertrophy extends. The color of the incised mass is grayish-yellow, resembling tissue which has undergone "lardaceous, fatty, or gelatiniform transformation," the bulk apparently being hypertrophied fibrous tissue.

There is said to be inflammatory obstruction of the lymphatic and vascular channels in the affected areas, supposedly produced, in the endemic type at least, by blocking of these channels by the *Filaria sanguinis hominis*. It is stated that mosquitoes are the carriers of the larva of the filaria; that the parent worm inhabits

the lymphatic trunks, and its ova are thence swept into the general circulation; that the greater the obstruction by the parent worm or its embryo, the more severe the elephantous symptoms. In many cases the *Streptococcus erysipelatis* is said to be responsible for the mischief, the disease being then an atypical erysipelas. In most instances, however, it is "obvious that the lymphatic system is chiefly involved, the lymph-vessels becoming tumid and tense, and, when punctured, giving exit to a chylous or milky discharge."

Elephantiasis may attack any part of the body; but the lower extremities are usually first involved, then the genital organs,—the penis and scrotum in males, the labia and clitoris in females. The elephantous enlargement may reach enormous proportions, as hereinafter shown.

In the endemic type, reports state there is a prodromic stage, characterized by febrile temperature, alternating with sweating, extreme distress, severe lumbar or articular pains, etc. This does not appear to be true of the sporadic type, at least four of the cases seen in Kentucky have not presented such symptoms, nor was there, so far as ascertainable, the enlargement of the bony structures which is said to occur in the endemic type.

The disease attacks both sexes,—most frequently the male,—the proportion being about four males to one female. Of five cases seen in Kentucky, four were males and one female. Dark races and persons living in unsanitary and unfavorable hygienic surroundings seem most susceptible to the affection. The cases observed here occurred in negroes, and all except two were hospital patients.

Morrow states that one of the striking features of the disease is its evolution by accesses; that after a febrile exacerbation with an increase of the tumefaction, with or without erysipelatous or lymphatic involvement, or inflammation of the veins, there usually follows an amelioration of symptoms; that sooner or later in typical cases there is a recurrence, sometimes in exaggerated type, of all the morbid phenomena. The same writer states that when the extremities are affected the disease is usually unilateral. In three of the cases seen here (and the Samoan is included in the series) the enlargement was bilateral, and in only one case was there a history of constitutional disturbance. The hypertrophy in all cases was marked, and there were no symptoms except those resulting from excessive enlargement of the affected parts.

Case I.—Negro, aged twenty years; native of Kentucky. Both legs involved to the knees only. History of constitutional disturbance,—*i.e.*, fever, sweating, and pneumonia (?),—three years previously, followed by inflammation and gradual enlargement of the feet and legs. When observed, the legs presented a typical elephantiasis, hypertrophy being extensive, the skin hanging in unsightly folds or wrinkles just above the ankles. When he passed from observation the disease had not extended above the knees. Measurement of left calf, twenty-four and a half inches; right calf, twenty-three inches.

Case II.—Negro, aged about twenty-five years; native of Louisiana; general health good. Extensive elephantous enlargement of both legs to the knees. Measurement, thirty-one and thirty inches respectively at the calf. When last seen, enlargement had not extended above the knees.

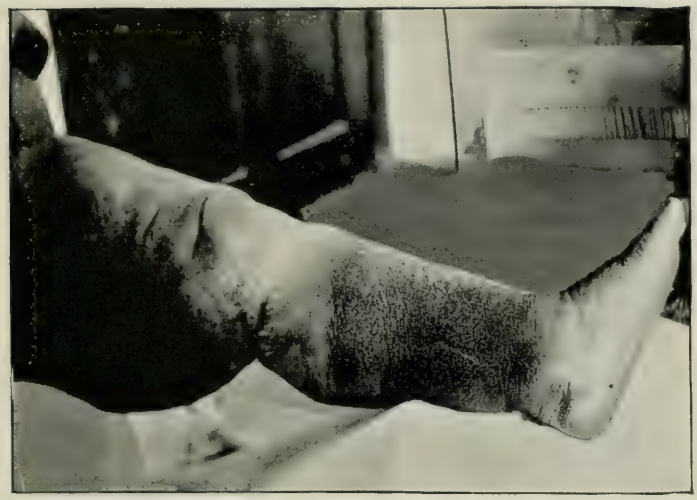
Case III.—Negro, aged thirty-three years; native of South Carolina; in good health. Enlargement unilateral; measurement, thirty-three inches at the calf. Amputation was performed above the knee by a Louisville surgeon. The thigh was not involved. When last seen, there was slight elephantous enlargement of the opposite leg.

Case IV.—Negro, aged twenty-eight years; previous condition and birthplace unknown. Unilateral enlargement involving entire leg. Measurement, thirty-two inches at calf. Amputation at the hip was performed by a Louisville surgeon. Patient died several weeks later from an intercurrent affection.

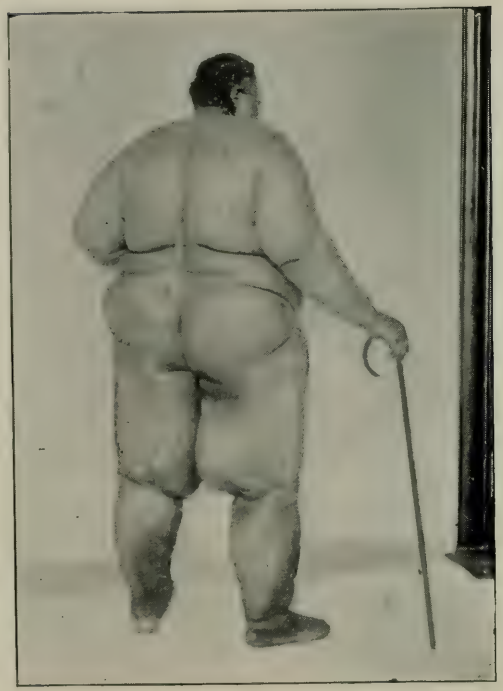
Case V.—Negress, aged fifty-two years; native of Kentucky; in good health. Elephantous enlargement involving almost the entire body. Height about five feet; weight, four hundred and sixty-eight pounds. She is still living, and remains well physically, notwithstanding the enormous hypertrophy, as shown by the accompanying illustrations. The writer is indebted to Dr. J. B. Marvin, of Louisville, for the photographs from which these illustrations were prepared. (Figs. 1, 2, and 3.)

Case VI.—A report of this case was published some four years ago,[1] the only obtainable information being a letter, which is reproduced in part, from a gentleman who spent many years on the

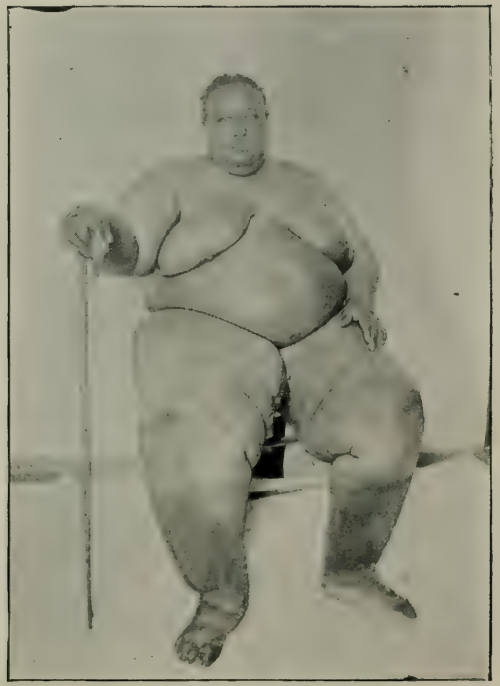
[1] Medical Times, October, 1895.



Case V.—Fig. 1.—Left leg.



CASE V.—FIG. 2.—Posterior view.



CASE V.—FIG. 3.—Anterior view.

Southern seas. The two illustrations are self-explanatory. He states: "In handing you the enclosed photographs a brief history elicited from the man himself, and a few ideas gleaned from observations and inquiries in Samoa, Africa, etc., may not be uninteresting. The man is a native of Apia (Upolu), Samoa (Polynesia). I first saw him in the sixties, while on a cruise to the island of Samoa, and met him also on several subsequent trips. He first noticed an inflammation and swelling of the lower extremities when fourteen years old, which persisted with periods of exacerbation and amelioration, without pain or other symptom, until the age of twenty-three, when the first photograph was taken. The progress that the disease had made from that time until the patient was forty-seven years of age, when the second picture was taken, will be readily observed. He was able to get about fairly well, although his ridiculous attempts at locomotion, carrying the enormous pendulous scrotum, can be better imagined than described. His general health was unimpaired; he ate and slept well, and all functions of the body were properly performed. He married at eighteen, and within the six succeeding years his wife gave birth to three healthy children. On my last trip to Samoa, the father, mother, and children were living, none of the latter having exhibited evidences of elephantiasis.

"Although it has been several years since I saw this man, elephantiasis has not died out in the Samoan Islands, as I have seen other cases on subsequent visits there and elsewhere in the tropics.

"I am told that elephantiasis invariably begins at the toes, thence making its way to the knees, later spreading to the thighs and forearms, finally affecting the scrotum and penis in the male, and the labia in the female. This was the course of the case.

"The mystery surrounding the disease seems to be exactly how it originates, and why it attacks certain individuals. It is not confined to males, but attacks females with greater or less frequency. It is said to be not hereditary, nor is it contagious.[1] There appears to be little or no odor from the affected parts, nor have I observed an abrasion or ulceration of the skin over the thickened area.

[1] Several cases of congenital elephantiasis, seemingly well authenticated, have recently been reported. See article by Dr. Moncorvo, of Rio de Janeiro, published in Pediatrics, December 1, 1897.

"Another feature sufficiently peculiar to be worthy of mention is that the disease in its progress never attacks a vital organ, nor have I known death to result directly from its effects. Persons having elephantiasis are otherwise in apparently good health, and only suffer the inconvenience of carrying about the enormous weight of hypertrophied tissue.

"The most marked case that I have seen in all my travels occurred in the person of an Ethiopian (Africa), aged seventy years, whose 'wool' was perfectly white, and who had been the subject of elephantiasis for upward of fifty years. His lower extremities had become enormous, and when standing erect the scrotum nearly touched the ground. (Figs. 4 and 5.)

"I have encountered the disease in the Samoan Islands, in the Caribbean islands, and in certain districts of Africa. I also understand it is quite common in the interior of China. It does not seem to be strictly climatic. As to its relative frequency, for instance, in the Samoan Islands, in a village of from two thousand to three thousand population there will often be found five or six cases of elephantiasis.

"The nature of the disease does not appear to be dropsical, aspiration revealing only the normal fluids,—blood, lymph, etc.,—and the enlargement is as firm and solid as other parts of the body, being simply an hypertrophy."

Other parts of the letter contain matters of interest, but for lack of space they are omitted.

An abstract of a case of sporadic elephantiasis recently seen in Memphis, Tennessee,[1] may not be devoid of interest.

"The accompanying illustration (Fig. 6) is from a photograph taken of a laborer who presented himself to the United States Examining Surgeons' Board for examination, alleging disease of the scrotum. His penis measured ten by twelve and a half inches, and his scrotum ten by thirty-six inches. He gave a history of cystitis following exposure. There were present cicatrices over the perineum, scrotum, and pubes. Urine was passing involuntarily from a fistulous opening in the upper anterior part of the scrotum.

[1] Elephantiasis of Scrotum, Dr. G. G. Buford, Memphis, Tennessee; Memphis Lancet, March, 1900. This abstract and the illustration are inserted with the kind permission of the author and publisher.

"The condition is rare in this climate, though common in tropical countries. Ablation is the only cure. Surgical interference was tendered in this case, but respectfully declined."

Dr. Buford further states that "elephantiasis" is the result of irritation, the legitimate sequence of which is hypernutrition."

The elephantiasic enlargement of the scrotum and penis is well shown in Figs. 4 and 5. While it is believed that the author may find it difficult to explain the pathological changes by his theory of "irritation," still his views are entitled to consideration, especially as the etiology of the disease remains practically unknown.

The cause of elephantiasis is more or less obscure, as evidenced by text-book statements,—viz., "lymphatic and vascular obstruction by the filaria," and infection by the *Streptococcus erysipelatis,* two distinctly different causative agents, both of which may or may not be concerned in the production of the disease. There is no history of these two agents acting in conjunction to produce the affection, nor have we the history that when one is present the other coexists.

If the filaria causes the supposed lymphatic and vascular obstruction, and if it be true that the germ is disseminated through the medium of mosquitoes, as claimed, why does the disease attack certain individuals, while others in the same family, living under the same sanitary and hygienic surroundings, and partaking of the same food, drink, etc., remain exempt? Susceptibility hardly explains the occurrence.

The sea-captain, whose letter is partially reproduced, states that the prevailing idea among the inhabitants of the Samoan Islands is that the disease results from sudden chilling of the feet and legs of the individual; that the filaria and the *Streptococcus erysipelatis* have naught to do with its causation; *e.g.,* the natives carry upon their backs heavy loads of natural products from plantation to market, and in so doing ford streams of cold water; being overheated from exertion, in fording such streams there is a sudden chilling of the feet and legs, and a consequent inflammatory reaction, resulting in interference with the vascular and lymphatic circulation, which conduces to the development of elephantiasis, if it does not constitute the sole cause of the disease.

It seems strange if marked vascular and lymphatic obstruction exists, which means diminution of nutrition and impairment of

vitality, that rapid hypertrophy of tissue should supervene. Reasoning *a priori*, accompanying such obstruction we should have serous and sanguineous effusion or extravasation into the subcutaneous structures, perchance death of the part *en masse*, gangrene, sloughing, etc. This, however, is not the case; therefore we must search elsewhere for the *fons et origo mali*. Again, if mosquitoes distribute the germs of filaria, and if these germs cause the disease, in what way do they gain entrance into the human system, and why do the vascular and lymphatic channels of the lower extremities, as a rule, first suffer? Moreover,—and this question has been propounded elsewhere and remains unanswered,[1]—why is there such a paucity of cases in districts where mosquitoes are abundant, and why does the disease develop in only certain individuals?

It is stated in text-books that ulceration, lymphorrhagia, and hemorrhage frequently occur when the extremities are involved in the morbific process. Observation shows this to be untrue in five sporadic cases; and where the disease occurs in the endemic type I am informed that the three conditions mentioned are practically unknown as concomitants, except as sequelæ to external violence, accidental or otherwise. In some cases, however, in which the disease affects the legs, the natural folds of the skin are so increased as to produce extensive linear depressions, resembling crevasses in the tissues, in which the natural secretions accumulate; these products undergo degenerative metamorphosis and give rise to malodors unless strict cleanliness is observed, and this fact may have given the impression that the tissues themselves were ulcerated. Warty or keloid growths, vegetations, eczema verrucosum, ulcerations, lymphorrhagia, and hemorrhages do not occur in elephantiasis, according to information possessed by the writer, which at best is limited.

The prognosis of elephantiasis is always grave as regards complete recovery, no matter what method of treatment is employed; but as to life the prognosis is favorable. Its chronicity is the most characteristic feature, and in the earlier ages its cure was seldom attempted; such cases were looked upon as hopeless from the beginning, and the duration of the disease was thought to depend upon the length of time the affected individual lived,—*i.e.*, until death

[1] Mapes, loc. cit.

resulted from other cause. Elephantiasis *per se* rarely destroys life, and the affected individual usually succumbs to some intercurrent malady.

As to treatment, a late writer (evidently referring to the endemic type) states: "During the febrile accesses of the disease, those which precede its earliest manifestations and those which so often accompany its evolution, the patient is to be given the usual cooling draughts and the antithermal remedies which have been found effective in the treatment of fevers in general. The hygienic management of these cases is of the highest importance, and often involves a change of climate and a residence where the disease is not endemic, and where also there is an absence of malaria, of much dampness, and of vitiated air.[1]

"The local treatment of advanced cases is by excision, with proper surgical precautions, of all the larger tumors. In this way growths of several hundred pounds' weight have been removed. In the case of genital tumefactions, the penis and testicles have been carefully dissected out from the sulci in which they were lodged, with the result, after cicatrization has been established, of leaving these organs unimpaired. Ligation of the larger vessels, as inducing still further obstruction of the vascular channels, is now practically abandoned as a therapeutic measure. The aid derived from compression with the elastic bandage is in most cases considerable. Persistent compression of the affected parts has been continuously employed for long periods of time with excellent results. Bandages for this purpose are preferably made of rubber, but have been effective when made of flannel cut on the bias, as has the ordinary roller bandage of the surgeon, applied over compresses. Methodical compression of the vessels supplying an elephantiasic mass has been successful in some cases, and in others followed by gangrene. Inunctions with mercurial, salicylated, tarry, and other medicated ointments are often indicated, and in some cases are useful. Alternations of hot and cold local baths, steam-

[1] From the author's statements in this paragraph it would appear that he has no definite conviction as to the causation of elephantiasis, first associating it with "fevers in general," then with "malaria," "dampness and vitiated air," and advising a "change of climate" as appropriate treatment.—C. C. M.

ing and showering the parts, massage, and the use of the constant electrical current have all been employed with advantage." [1]

When the disease has progressed until considerable enlargement of the extremities has taken place, nothing can be gained by temporizing; excision of the limb, when the affection is unilateral, is indicated if hope for a permanent cure is to be entertained, and even then the opposite leg will more than likely be similarly involved sooner or later. When the genital structures of the male have assumed enormous proportions, as in Case VI., excision may be advisable,—not to cure the disease, but to relieve the patient of his burden, thus facilitating locomotion and conducing to comfort.

In the earlier stages, if the disease could be differentiated from other inflammatory conditions,—*i.e.,* prior to any considerable enlargement of the affected parts,—much benefit might be derived from continuous pressure by appropriate means. When the disease has become fully established, after the morbific condition has become unmistakably manifest, after considerable hypertrophy has occurred, the writer fails to understand how "change of climate" could produce material benefit. If the causative agent is as stated (the filaria), when once implanted into the human system, is there reason to believe that change of climate will eradicate or destroy it? If, on the other hand, the claims of authorities as to causation are erroneous (which is not improbable), and if the disease is the result of some general systemic condition, then benefit might follow a radical change of climate and marked improvement in sanitary and hygienic environments. Again, if the disease be due to sudden chilling of the feet and legs, where these parts are first involved, explanation of causation is still wanting where other parts of the body are first attacked, and where the disease occurs as a congenital malady.

At all events, after the disease has progressed to the state of marked hypertrophy of the parts involved, little benefit seems to have followed general treatment, and *in rerum natura* we do not see how the filaria, granting this to be the causative agent, could be dislodged, and the vascular and lymphatic circulation restored, by systemic medication.

[1] To form conclusions as to the extremely unsatisfactory and unsuccessful treatment of a given disease, one has only to note the almost innumerable remedies and methods suggested for its "proper management."—C. C. M.

The curability of elephantiasis, as already suggested, was seriously questioned by ancient writers, which view is also held by some more modern observers, though, as shown, if it were possible to recognize the disease in its incipiency, if there were any pathognomonic symptoms in the manifestations of the so-called prodromic stage, then by the adoption of adequate treatment,—*i.e.*, by the application of persistent compression and the administration of appropriate systemic remedies,—the disease might be arrested; but in many cases (at least in the sporadic type) there occur no symptoms by which the malady may be recognized with any degree of certainty until marked enlargement of the affected parts supervenes, and, this being true, relief cannot be promised except by the adoption and rational application of modern scientific surgical principles.

Where the elephantous enlargement affects the entire body, as in Case V., surgical treatment is, of course, contraindicated; and as such patients usually enjoy good general health, and as nothing in the way of internal medication has proved of more than questionable benefit, no treatment is necessary except that directed to relief of pressure symptoms should such occur.

THE IODIDE TREATMENT OF ANEURISM.

LECTURE DELIVERED BEFORE THE SECTION ON PRACTICE OF MEDICINE, NEW YORK ACADEMY OF MEDICINE.

BY LOUIS FAUGÈRES BISHOP, A.M., M.D.,

Assistant Attending Physician to the French Hospital; Attending Physician to the Colored Hospital; late Chairman of the Section on Practice of Medicine, New York Academy of Medicine.

Gentlemen,—I have been assigned to the defence, as it were, of a well-intrenched, long-maintained base, while others will reach out to establish new positions. To them may be attached greater interest, but it is ever important that bases should be well defended. I feel the more like doing this because I find so often that physicians neglect many useful procedures in their desire to test something new.

I have no cases of aneurism cured by the use of potassium iodide to report, but I have seen so large a number in which the condition was ameliorated that I am a firm believer in its usefulness. Most of my cases have indeed been such that the expectation of cure was not to be entertained. The administration of potassium iodide in twenty-grain doses three times in the twenty-four hours was initiated as soon as the cases had been properly examined, and was maintained over long periods of time. The immediate effect of the drug was that the patients were made more comfortable. The pulsations of the sac were less and its size apparently diminished. On this treatment some of these patients lived a long time without very great suffering. The effect of the iodide cannot be said to be fully explained by the theoretic diminution of blood-pressure. It seems to have a specific influence upon the sac itself, whereby its development is retarded and coagulation favored.

An increase in the rapidity of the heart's action is a symptom of iodism. If preferred, sodium iodide may be used instead of the potassium salt; and there are therapeutic reasons why it is preferable for use for a long period of time. The iodide treatment

has at least this to recommend it in preference to other treatments, that it is directed to the tissue-changes that were the original cause of the disease. Whatever else the iodide may accomplish in the body, it certainly has a greater influence over tissue-metabolism than any other drug; and it may easily be surmised that the iodide might in many cases, particularly those of specific origin, prevent the formation of aneurism in an early stage. All other methods of treatment by inducing coagulation have this weak point, that they seek to cure by a natural process a disease the causes of which are still active.

The iodide, as in other conditions, should be commenced with doses of ten grains three times a day, and increased gradually up to the maximum dose. Our practice has been to follow the custom of ordering one grain a day added to each dose, so that on the second day the patient would get eleven drops of the saturated solution in the morning, the next day in the morning and at noon, and so on. If there are signs of iodism, the iodide is stopped, and later resumed in doses of, say, five grains, and then gradually increased.

Another strong argument for the use of the iodide, but one which does not wholly explain its effect, is that aneurism is often seen in syphilitic subjects. My plea is for the use of the iodide in all cases of aneurism whether undergoing other treatment or not. It does not interfere in any way with local mechanical treatment, nor, I believe, with the use of other agents.

In these remarks I particularly refer to thoracic aneurism, because without doubt surgery, whenever applicable, offers the best hope of cure. Whatever may be the reports of any treatment, it will be hard to credit the permanent cure of aneurism opening by a large orifice from the aorta near the heart.

I know that in purely scientific investigation it is unsatisfactory to mix issues, but in formulating the treatment of a case of thoracic aneurism I would say that the use of the iodide and all other means available should be persistent.

ATYPICAL ENTERIC FEVER.

CLINICAL LECTURE DELIVERED AT THE PENNSYLVANIA HOSPITAL.

BY J. C. WILSON, M.D.,

Attending Physician to the Pennsylvania Hospital, Philadelphia.

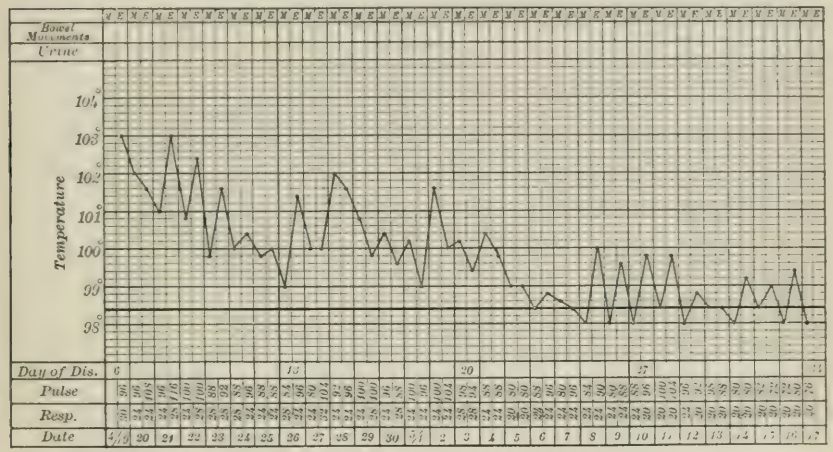
GENTLEMEN,—The woman in the private room, Station B, whom you saw some time ago suffering from fever, is now fully convalescent and has been allowed to return to her home.

Had the case been one of ordinary enteric fever, such as is frequently demonstrated to you in the amphitheatre of the hospital, and such as may be seen every day in the wards, I should not have regarded it as worth our while to review it at this time. Nor should I have done so had it presented the more familiar complications or sequels of the disease. But certain phenomena attending the onset and early course of the attack, not usual in enteric fever, others developing at a later period, and, finally, the absence of common symptoms, tended to obscure the true nature of the disease and render the diagnosis uncertain.

The patient, an unmarried German woman, thirty years of age, a teacher, was admitted April 18, 1900. She stated that she had occasionally suffered from attacks of indigestion and pains referred to the distribution of the left sciatic nerve. Otherwise her health had been excellent.

About two weeks before admission she had pain in the legs, especially in the left, and felt weak. She was, however, able to go about and attend to her duties, which left her much more fatigued than usual at the end of the day. She expressly states that she had no headache during this time. Six days prior to admission she was very chilly during the evening, began to suffer from headache and dizziness, and had fever.

The following day she was obliged to remain in bed. She summoned a physician, who found her temperature in the afternoon to be 105° F. From this time the temperature ranged for several days between 104° F. and 105° F. There was great weakness, together with continuous free sweating and constipation. The administration of a laxative produced one or two loose movements, but nothing like diarrhœa. There was nocturnal delirium, with great restlessness and mental agitation. During the day the patient was nervous and depressed, but free from delirium.



Temperature chart of Dr. Wilson's case of atypical enteric fever.

On the sixth day after the beginning of the attack I saw the patient in consultation with her physician. Her temperature was then 104° F. She was bathed in profuse perspiration; her tongue was moist, and coated with a thick yellowish-white fur. There was no desire for food, and the bowels were constipated. The patient complained of pains in her limbs and back of the eyeballs, but was free from headache. She was very nervous. The most striking phenomenon at this time was a moderately coarse tremor of the eyelids when closed, of the tongue, lips and muscles around the mouth, and of the hands. No rose spots or other eruption were discovered. There had been no epistaxis. That afternoon she was sent to the hospital.

The following note was made by the resident physician. Pa-

tient seems confused, has difficulty in concentrating her thoughts, is restless and agitated. Pupils not dilated and respond normally to light. Tongue moist, thickly furred, and trembling. Temperature 103° F., pulse 96, respirations 30. Heart sounds of normal intensity; no murmur. Physical examination of the lungs gives no abnormal signs. Area of liver dulness not increased; area of splenic dulness distinctly enlarged. Abdomen not distended, walls soft, no tenderness.

On the day after admission two rose-colored spots were found, more elevated than the lenticular enteric fever spots and not wholly disappearing when the skin was made tense. These spots disappeared in a few days and no others were noticed during the course of the disease. The urine was normal.

The temperature range is shown in the accompanying chart. It is extremely irregular and does not conform to the usual type.

The defervescence was completed about the twenty-second day of the disease. Later there developed oscillations between 98° F. in the morning and 100° F. in the evening, which ceased when the patient was first allowed to sit up, upon the twenty-ninth day of the attack.

For about a week after admission there was mild wandering with uneasy delirium during the day and great agitation at night. For ten days from the time of admission catheterization was necessary. Constipation continued, making the occasional administration of enemata necessary.

A few days after admission the patient for the first time complained of pain in the back of her head and in the neck, extending down towards the left shoulder. A day or two later movement of the head became extremely painful; the patient lay with the face turned towards the right and the head somewhat retracted, complaining of great pain on any attempt at movement. In a week these symptoms began to subside and a little later wholly disappeared.

The treatment was symptomatic. It consisted of the administration of sodium bromide, hypodermic injections of morphine and hyoscine hydrobromate, cold sponging and ice-caps to the head and neck. Later dilute hydrochloric acid and quinine were administered. The diet consisted of milk and lime-water, broths, and raw eggs. No solid food was given until after the deferves-

cence. The craving for food, which is more urgent in the early stage of convalescence from enteric fever than in other common diseases, became marked as the temperature reached normal.

The Widal test yielded negative results upon the eighth and upon the fifteenth days, and a positive result upon the thirty-first day of the attack. These three observations only were made. It is to be regretted that the test was not repeated at more frequent intervals. The late positive reaction, however, rendered the provisional diagnosis of enteric fever positive.

In this connection I must remind you that the agglutinating property is not acquired by the serum until after the disease has made some progress. Practically positive results are not common until the middle of the second week; very often not until later than this. In a small percentage of the cases a positive reaction has not been obtained during the patient's stay in the hospital; that is to say, at any time throughout the period of convalescence. Thus, in five instances in the records of one hundred and forty-seven cases among soldiers which I studied in the German Hospital, Philadelphia, during the great epidemic of 1898, the results as shown by the hospital laboratory and the city laboratory were wholly negative. Four of these cases were unquestionably enteric fever, while the fifth was doubtful.

This case, then, must be looked upon as one of atypical typhoid fever. Cases of this kind throw valuable side-lights upon our knowledge of the infectious diseases. They are difficult of diagnosis and test the resources of the practitioner to a high degree. The vacillating physician who in private practice makes in such cases a new and less positive diagnosis from day to day strains the confidence of the patient's friends. From this point of view the atypical cases of enteric fever may be grouped with the *opprobria medicinæ*. It is a good rule of practice to decline to make a diagnosis until the facts justify it. To the student, however, who has familiarized himself with the text-book descriptions of disease and who has had some opportunity for clinical observation, such cases are most instructive. The departures from type emphasize the importance of its recognition; aberrance by antithesis implies a sequence of morbid events according to rule. To observe that there are atypical forms of disease widens the horizon in regard to the nature of infection. Variations in the intensity of the in-

fective principle fall within the scope of the pathologist and bacteriologist rather than of the clinician. Variations in the reaction on the part of the individual infected modify the clinical picture. Hence different groups of the infectious diseases, as fulgurant, ordinary, larval, and the like; or, when the reaction is conspicuous on the part of a certain physiological system, modifications expressed by such terms as cerebral, pulmonary, cardiac, gastro-intestinal. Very often, as in the case under consideration, the departures from the type cannot be explained.

The difficulties in this case related wholly to the diagnosis. The treatment throughout was symptomatic. At the beginning of the attack the disease was thought to be influenza. About the fifth or sixth day a provisional diagnosis of enteric fever was made. Some days later a question arose as to cerebro-spinal fever, and finally continued malarial fever had to be considered. Let us take these up in turn, having due regard to the temperament of the patient, who described herself as an overworked teacher of intensely nervous organization, and who said, " We Germans are regarded as phlegmatic, but when that condition gives way we are as nervous as Americans."

1. *Influenza.*—The diagnosis of influenza of the nervous form then prevalent was justified by the sudden onset with chilliness, pains back of the eyes and in the limbs, great prostration, high temperature, and continuous sweating. The nocturnal delirium and the remarkable tremor were not against this view. By the end of a week, however, the persistence of the symptoms discredited the diagnosis of influenza,—a disease which in uncomplicated cases rarely exceeds from five to eight days in duration.

2. *Enteric Fever.*—This provisional diagnosis was made about the sixth day by exclusion. There are very few acute febrile diseases other than enteric fever encountered in this climate which do not show by the end of the first week local manifestations. In this case, at that period there were none save enlargement of the spleen, a sign in itself in favor of enteric fever. About this time two rose-colored spots appeared in the epigastric zone to the left of the median line, not wholly disappearing on pressure and more distinctly papular than the lenticular rash of enteric fever usually is. The agglutination test also failed and the diagnosis remained uncertain, especially as the abdomen was soft, slightly retracted rather than tympanitic, and everywhere free from tenderness.

The temperature chart did not help us. When the patient first came under medical observation the day following the chill and, according to her own view, the second day of her illness, the evening temperature was 105° F. For some days it ranged between 104° F. and 105° F., but upon the sixth day of the attack it fell to 103° F., and from this point by a protracted lysis of great irregularity fell to normal on the twenty-second or twenty-third day,—a range wholly unlike that of the so-called typical enteric fever temperature which Wunderlich originally described and which serves as the basis of the descriptive accounts in the text-books. Nor did the agglutination test again made with a negative result upon the fifteenth day of the attack support the provisional diagnosis. Constipation and retention of urine continued, and the spleen remained moderately enlarged. Meanwhile a new set of symptoms developed which were very suggestive and added still further to the diagnostic uncertainties.

3. *Cerebro-Spinal Fever.*—Towards the close of the first week after admission the patient, who had not had headache, began to complain of pain in the back of her head and neck, extending downward towards the left shoulder. This pain was constant and severe; it was accompanied by tenderness upon pressure, and there was found to be rigidity of the muscles. A slight degree of opisthotonos developed with rotation of the face to the right. Movement was impossible on account of pain. Headache in the ordinary sense was evidently not present. There were no pupillary phenomena, no herpes, no petechiæ. There was complete loss of appetite, but no vomiting. The deep and superficial reflexes were normal.

Hot-water bags and subsequently a blister were applied to the nape of the neck. In the course of a week these symptoms subsided and presently wholly disappeared.

The occurrence of this group of symptoms suggested cerebro-spinal fever,—a possibility in the case not lightly to be passed over, since sporadic cases of cerebro-spinal fever had recently occurred in Philadelphia, and a case of the kind was at that time in the women's ward of the hospital. Other symptoms in the case seemed for a time to lend support to this view, which we carefully discussed, but did not adopt. These were the sudden onset of the attack, the high fever in the beginning, the irregularity of the temperature

curve later, the moderate enlargement of the spleen, the absence of intestinal symptoms, intense nocturnal delirium, sweating, and the weakness and rapid wasting which now appeared. It is true that the familiar attendant symptoms of the initial stage of cerebro-spinal fever,—namely, headache, vomiting, and painful retraction of the back of the neck,—were not present, nor was Kernig's sign elicited; but those who have had the opportunity of observing cases of cerebro-spinal fever in epidemics know that there are atypical cases of this disease also, and that the range of modification is perhaps greater than in any other of the acute infectious processes. Stillé, speaking of this peculiarity of cerebro-spinal fever, called it the "chameleon-like disease," and said of the larval cases that they were only to be recognized in the "lurid light of the prevailing epidemic."

The absence of headache is important in this case, but there are individuals who do not know what headache is, and this patient stated that she had rarely suffered from it.

There are cases of enteric fever, as you well know, that present during their early course a resemblance to cerebro-spinal fever so close that the differential diagnosis cannot be made. This group of cases has been described as the cerebro-spinal form of enteric fever. Again, cerebro-spinal meningitis may arise during the course of an attack of enteric fever from secondary infection, the onset of the complication under these circumstances being almost always insidious, as its result is almost invariably fatal. These points were carefully considered, without being permitted, however, to modify our provisional diagnosis of enteric fever.

The question of lumbar puncture for diagnostic purposes naturally arose. It was settled adversely, chiefly because of the extremely excitable and nervous condition of the patient, and also because the early and progressive amelioration of this set of symptoms rendered it unjustifiable.

As the symptoms of which I have been speaking subsided, another set of clinical phenomena appeared which demanded consideration.

4. *Malarial Fever.*—About the sixteenth day of the attack the patient began to complain of attacks of chilliness in the early afternoon followed by sensations of discomfort, flushing of the face, and a sense of fulness in the head and slight sweating. Oscilla-

tions of temperature not recorded upon the morning and evening chart, but shown upon the chart of three-hour temperatures, now appeared. For five days the temperature rose with great regularity about four o'clock to 103° F., and this rise of temperature came between the period of chilliness and that of sweating. The blood, which had been previously examined for the malarial parasite with negative results, was carefully re-examined, but no hæmatozoa were discovered; nor was there leucocytosis. The patient was nevertheless given quinine in large doses, and at the end of five days the periodical paroxysms of chilliness, the mid-afternoon elevation of temperature, and the sweating ceased.

There are those who with ample opportunities for observation do not believe that malarial fever of irregular, or atypical, types occurs in Philadelphia. I do not share in this belief. There is now in my service in the women's ward of the hospital a patient with chronic malaria, in whose blood numerous tertian parasites may be seen, but who has neither distinct ague paroxysms nor elevation of temperature. Such cases are far from rare, and it is therefore our rule in this hospital to examine the blood for the malarial organisms in all cases of protracted ill health without clinical phenomena adequate to account for it.

Nor must we overlook the fact that according to the latest views in regard to malarial infection the vernal outbreaks are of the nature of relapses, and that the summer and autumnal cases only are due to fresh infection. Furthermore, it is not uncommon for a latent malarial infection to show itself late in the course of, or during convalescence from, an attack of enteric fever. Nevertheless, the original provisional diagnosis still seemed the probable one. This view was fully confirmed upon the examination of the blood-serum for the agglutination test on the thirty-first day of the attack. The result, as has been stated, was positive, and the true nature of the case was established.

It is interesting, I trust, to you as students of clinical medicine to have thus passed in review the difficulties of this case. Even now, after the disease has run its course and the patient is well, these difficulties vanish only in part. To affirm that they are due to peculiarities in the reaction of the individual to the enteric fever infection is admissible, but it is not an explanation.

Suddenness of onset is by no means uncommon in enteric fever,

and it is most likely to occur in those whose nervous equilibrium is unstable,—namely, children and neurasthenic and hysterical adults. Tremor and violent nocturnal delirium occur in individuals of the class just named. Very high temperature at the onset may be attributed to the same cause. Violent oscillations of temperature suggestive of ague paroxysms and followed by sweating are not so readily to be accounted for. Nevertheless, it is possible that they may have been manifestations of the reaction of a very unstable nervous system to the enteric fever infection. The pain, tenderness, and spastic rigidity of the neck were probably the exaggerated manifestations of a transient occipital neuralgia. All things considered, the study of this case has suggested many thoughts that may be of use to you.

Neurology

LOCOMOTOR ATAXIA.

CLINICAL LECTURE DELIVERED AT THE CHICAGO POLICLINIC.

BY HUGH T. PATRICK, M.D.,

Professor of Neurology in the Chicago Policlinic; Associate Professor of Nervous Diseases in the Northwestern University Medical School; Neurologist to the German and Maurice Porter Hospitals, etc.

GENTLEMEN,—The first statement that I wish to make is that in the recognition of the *disease* locomotor ataxia the *symptom* locomotor ataxia is of very minor importance. There is a disease which is called locomotor ataxia by virtue of consensus of medical men; there is also a symptom locomotor ataxia which exists by virtue of physiology and pathology. Locomotor atxia, then, is primarily a symptom, and is incoördination in the act of progression or locomotion, and the disease locomotor ataxia was so named by Duchenne, of Boulogne, forty years ago in consequence of this symptom. Let me repeat, that in the recognition of the disease which by common consent we call locomotor ataxia, or tabes dorsalis (or tabes, for short), the symptom of incoördination in locomotion or in the act of voluntary movement is of very minor importance. I make this statement because the average physician, possibly owing to his early teaching, attaches altogether too much importance to the symptom of incoördination, and in consequence fails to recognize the disease in its earlier stages. Perhaps the best way to emphasize this dogma is by the presentation of a few cases.

CASE I.—This gentleman is fifty-five years of age, and his only complaint is that he cannot see. He does not complain of ataxia or of incoördination, but of loss of sight; yet it is a case of tabes. His occupation is that of newspaper seller. He stands for hours at a time without the least trouble; he can walk as well as a blind man

is expected to walk. Aside from a certain element of caution in his gait, there is nothing abnormal to be seen. You will notice that when he stands with heels and toes together (being blind, he does not need to close his eyes) his station is perfect. He has not the symptom locomotor ataxia at all, but he has the disease.

Case II.—Here is another man, kindly sent to me by Dr. Wilder, fifty-one years of age, whose vision began to fail six years ago. The probability is that failure of vision in the right eye began considerably earlier, although he was unaware of it, because when he first noticed that vision was poor and came to be tested, vision in the right eye was already very much diminished. That is all he complains of. He is a carpenter by trade, and he never noticed any incoördination. He still does manual labor, principally gardening, and his coördination, as you will see, is apparently perfect. The only condition that interferes with his usefulness is lack of sight. (Patient stands with his heels and toes close together, closes his eyes, and there is only a faint trace of swaying. One would not notice it if it were not carefully tested for.) This is another striking example of the fact that ataxia is not by any means the most prominent symptom in tabes. Indeed, it is the least prominent, for if we were to wait in either of these cases for the appearance of incoördination in order to make the diagnosis of tabes, we would have to wait for years. The probability is that neither of these patients, as we shall presently see, will ever develop much ataxia.

Case III.—The next patient I show you is a man thirty-five years of age. He was referred to me by Dr. Gradle, whom he consulted for trouble with his eyes. He sees double because of an almost complete paralysis of the third nerve on the left side. This is the striking symptom in his case. When he looks upward the right eye rolls up, while the left does not; when he looks down the right eye moves downward, but the left does not; when he looks to the right the left eye does not move beyond the middle line; he has a slight drooping of the upper lid on the left side, and that is about all he complains of. He does not complain of his legs. He has not, however, perfect coördination. In this case the principal trouble relates to the ocular muscles, but the disease is the same. When we ask the patient to put his heels and toes together, and to close his eyes, you notice a little swaying.

Case IV. was sent to me some months ago for sciatica and

chronic neuralgia. I asked him at the time how long he had had sciatica, and he replied, "Twenty-five years." I then asked him whether he had it all the time, and he said, "No; it comes in spells. The pains are terrible; I can hardly stand them." I then asked him how long these attacks of sciatica last when they appear. He said, "Sometimes an hour; at other times three or four days. They come and go suddenly." I said immediately to those present that this was not a case of sciatica, but in all probability one of locomotor ataxia. Examination proved this surmise to be correct, and yet, so far as I can learn, a correct diagnosis had never been made. For twenty-five years he has been suffering from the lightning pains of tabes. He had no incoördination at that time, and has only a slight trace of it to-day,—imperceptible if not carefully examined for.

Case V., a man of thirty-three years, came complaining of loss of sexual power, which began to diminish notably two years before. At first this was, so far as he noticed, his only disorder; but of late other symptoms, including some ataxia, have been added, but they are not sufficient to interfere with his very active life as a business man, largely on the road. If he were sexually vigorous, he would not be consulting a physician to-day. His sexual organs are normal, but his spinal cord is not.

Case VI.—This man asks relief from only one difficulty—what he calls "kidney trouble." What annoys him is frequent and urgent micturition, and if he does not immediately answer each call he regrets it to the extent of damp trousers. He says that if this trouble were remedied he would be entirely well, with the exception of some epigastric pain. Even as it is, he works hard all day as a machinist. His disease is tabes; he has a large amount of residual urine, but he has no incoördination.

I show you these cases for the purpose of emphasizing the statement I have just made, that in recognizing the disease tabes we cannot rely upon the symptom locomotor ataxia, for although in advanced cases the incoördination becomes a very prominent—possibly the most prominent—symptom, early in the disease it is uniformly absent. Incoördination is *never* the first symptom in tabes. *Never!* Then how are we to recognize the disease if we are not to do so by incoördination, and for what do the patients first consult a physician if not for trouble with their legs, or incoördina-

FIG. 5.—Method of performing flexion in a case of locomotor ataxia.

portion of patients come complaining of an uncomfortable feeling in the rectum, of rectal tenesmus, with sudden calls to evacuate the bowels. Sometimes they evacuate too promptly, at other times there is difficulty of evacuation when they once get to stool. There is a more or less constant feeling of discomfort about the anus or perineum. They say they feel as if something were in the bowel, or they complain of numbness. In such cases one should always examine for tabes.

Perhaps a smaller proportion of patients come first complaining of loss of sexual power (Case V.), and they have generally diminished desire along with diminished power. They complain that erection is imperfect, that they cannot have intercourse as frequently as formerly, and that ejaculation is premature. Sometimes they have a normal erection; usually they have not. In all such cases examine for locomotor ataxia, although the most of them will be found to be due not to tabes, but to some functional trouble. The greater number of cases of impotence are of psychic or functional origin, generally due to the mental state of the patient and not to locomotor ataxia; still, tabes must be examined for.

Many tabetic patients come to us complaining that the legs tire easily; they do not say that they are uncertain in their walk, but that their legs are easily tired, and that occasionally there is a tendency to drop suddenly as if some one had struck them behind the knees. In such cases always examine for tabes. A few cases first notice the disease by reason of failure in hearing. This number is not great, but there are a few; and very occasionally an ataxic becomes a patient because of a sluggish ulcer on the foot—generally in connection with a corn or callus.

Aside from the typical lancinating pains of tabes, which are idiosyncratic, unlike anything else, many tabetics complain of pains and paræsthesiæ which do not belong to this category. Of these, quite a number will fall, on careful examination, under the class of girdle-pains; but in inquiring for this symptom be careful not to say simply, "Have you a girdle sensation?" because the patient does not recognize it as such. It were better to ask if he has pains in the chest, or neuralgia in any part of the body, or pains in the stomach, etc. The gentleman here, who says he has had sciatica for twenty-five years, complains sometimes of a severe pain in his ribs. He has been treated for chronic intercostal neuralgia and for dys-

pepsia. The girdle sensation is generally a pain, and unless you inquire for pain you will not get what you are after. Other patients have pains in the legs, but they cannot be described as lancinating or shooting. They are largely aches. The tired feeling may be so intense that it amounts to a pain. There may be a feeling of numbness and of tingling, or a feeling as if there were something under the feet; and there is frequently numbness along the ulnar border of the forearm and in the last two fingers. These are the principal symptoms of which patients complain when they come to us with incipient locomotor ataxia.

What are the symptoms upon which we depend for a diagnosis of tabes? To use a Hibernianism, the first symptom is two; at least, it is bicephalic, and I cannot decide which is the more important. I will write upon the black-board the symptoms in what has seemed to me the order of their importance, and as we proceed you will notice that incoördination comes very low down in the scale.

1. *Loss of the Knee-Jerk.* 2. *Argyll-Robertson Pupil.*—These are the earliest and most important symptoms, because they are the most frequent, are very rarely found together in any other disease, and are purely objective. We can examine for these two symptoms, ignoring entirely the feelings, the opinions, and the emotional state of our patient. The reason why I have put the loss of the knee-jerk first is because it is the easier of the two to remember, and, once known, it helps you to remember what the other is. Of course, you all are aware that when light shines into the eye the iris contracts. To state it differently, when the retina is struck by a beam of light there is contraction of the pupil, just as there is contraction of the quadriceps extensor when the patellar tendon is struck with a percussion hammer or the finger. In tabes these reflex contractions are lost, but the pupil still contracts to accommodation, just as the quadriceps still contracts to volition,—to accommodate the patient, if you like.

If I were now asked to condense all that I have said into one sentence, I could do it by saying, Examine the knee-jerks and the pupillary reactions in every patient who comes to you. It takes but a moment, and you make yourself safe. A patient who has both absolutely normal is not a tabetic.

3. Next to these two symptoms I would place *bladder disturbance,* which may be of two kinds: the patient cannot urinate

promptly when he wishes, or he urinates too promptly,—he has a call to urinate, and before he reaches the urinal loses a few drops of urine in his trousers. Let us see what our patients show of these three symptoms.

The old gentleman has the Argyll-Robertson pupil complete. He has a very little knee-jerk, obtainable only with reinforcement, and occasionally passes a few drops of urine in his trousers. The next patient has no knee-jerk, but his pupils respond very faintly to light. They contract perfectly with accommodation; in other words, he has incomplete Argyll-Robertson pupils. The third patient has no knee-jerk; but he too has slight contraction of one pupil to light, the other iris being immobile by reason of paralysis of the third nerve. He has, then, beginning Argyll-Robertson pupil. He also has trouble with the bladder. Sometimes before he can reach the closet he involuntarily voids considerable urine. The fourth patient has loss of the knee-jerk, Argyll-Robertson pupils, and both the relative incontinence and relative retention of urine. Case V. has loss of the knee-jerk, Argyll-Robertson pupils, and impaired bladder control; and Case VI. has loss of knee-jerk and pupillary reflex to light, although he complains only of vesical insufficiency. The slight bladder disturbances are frequently referred to supposititious cystitis, urethritis, or enlarged prostate, or are overlooked entirely in our examinations, and yet vesical disturbance is of such great importance that I have put it down as the symptom third in importance in the diagnosis.

4. Next in weight I would place a history of the typical *lancinating pains.* They are very frequent in locomotor ataxia, and often precede by a number of years the other symptoms. For instance, in one of our patients (Case IV.) they have been present for twenty-five years, and yet he has no other striking symptoms of tabes. Another patient (Case II.) has had very sharp pains in his legs for the last four years, and yet there is no uncertainty in walking. Still another (Case III.) had these lancinating pains for two years before incoördination was noticed. Cases V. and VI. have had no lancinating pains.

5. The fifth symptom I shall put down is *analgesia of the legs,* meaning by that, of course, loss or impairment of the pain sense. In many of these patients sensation to touch is perfect, yet they have impairment of the pain sense below the knee. I have just

stuck a pin half its length into this patient's leg without causing pain, yet he feels the slightest touch of a pledget of fine cotton or a camel's-hair brush. The man who is nearly blind has not much interference with pain sense, but he has some. A third patient, when tested for pain sense, shows no abnormality. The fourth patient has complete loss of pain sense below the knee, but he also has some slight tactile anæsthesia. Cases V. and VI. show marked discrepancy between the tactile and pain senses.

6. *Previous Specific Infection.*—The value of such a history in the diagnosis of tabes is a matter of discussion, but in my opinion there can be no doubt that a history of syphilis is of considerable value. In private practice, where I am best able to get historic details, specific infection has occurred in a very large percentage of tabetic cases, and I believe this is the experience of most careful observers who obtain their information under favorable circumstances. So I consider it always necessary to inquire whether in the past there has been specific infection, or whether there has been anything in the history that can be construed into a diagnosis of syphilis. The time at which the specific infection has taken place, supposing it to have occurred, is of paramount importance. Syphilis is found to have existed in very many cases of tabes, but not recently,—that is, the chancre antedates the appearance of tabes by a number of years, generally from eight to ten or twelve. If the patient has contracted syphilis within two or three years, the probability is against his trouble being locomotor ataxia.

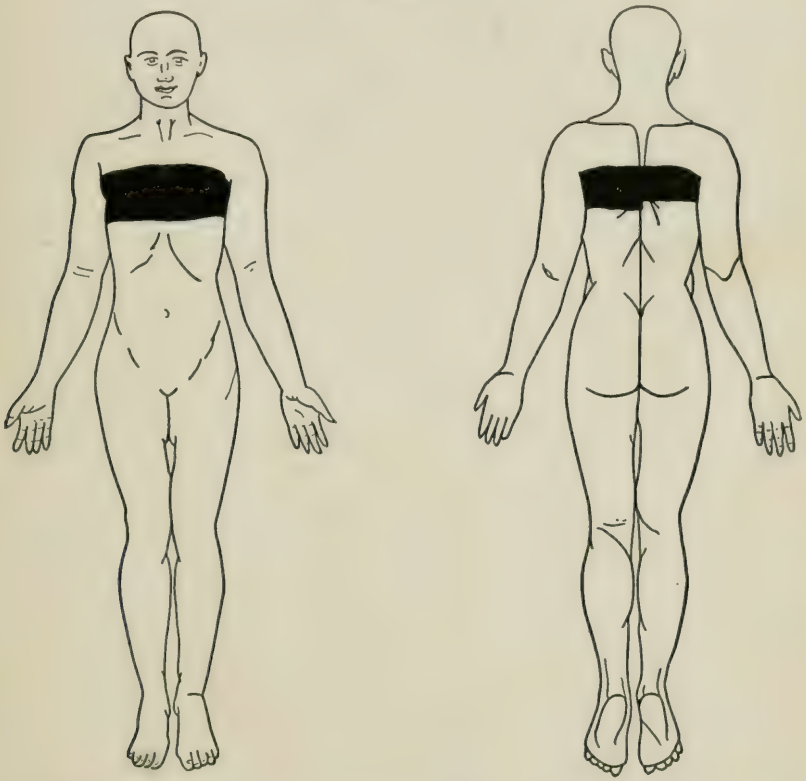
7. *Presence or History of Ocular Paralyses or Pareses.*—When paralysis of ocular muscles comes on suddenly or rapidly in an adult, the chances are easily nine in ten that it is due to one of three things,—tabes, brain syphilis, or general paresis; and of these, tabes is the most fruitful cause. A peculiarity about the ocular pareses of tabes is that they are likely to be transient. They come on suddenly, and in a week or two, or in a month or two, they disappear,—not always, however; sometimes they are permanent.

8. *Atypical Pains and Paræsthesiæ.*—As ranking next, we will name the atypical pains and paræsthesiæ,—the pains around the chest, the aching, tired feeling and numbness of which I have spoken. They are all important, although very similar pains and paræsthesiæ may occur in other cord diseases, in connection with other symptoms. For instance, although Cases V. and VI. have no

typical pains, the former has occasional sharp, stinging pains referred to the testicles, and the latter complains bitterly of frequent "stomach-ache," which is simply a tabetic pain referred to the epigastrium.

9. *Failure of Vision.*—In tabes this occurs early from optic atrophy; and when it takes place the other symptoms are in abeyance. This tardiness, or default, of other symptoms in the

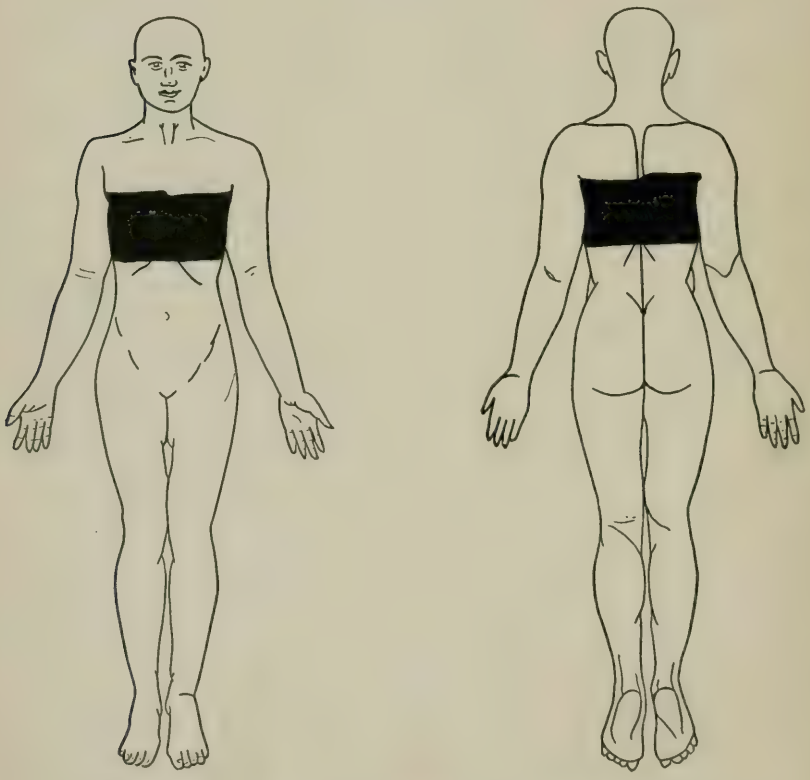
Fig. 2.



presence of optic atrophy led Dejerine to apply to such cases the inaccurate but graphically descriptive term "tabes arrested by blindness." If a patient comes to you with failure of vision from locomotor ataxia, you are justified in assuring that patient that he will always have good use of his legs. Similarly, if a patient comes to you with tabes and has marked incoördination, you may be sure that he will always have his vision. This is not a trivial

matter, because one patient consulting us for trouble with his legs may know of another patient with locomotor ataxia who has become blind, and *vice versa,* and he is greatly worried for fear he may yet acquire the additional symptom. It rarely happens in tabes that a man has marked incoördination with almost complete blindness. He may have one or the other, but very rarely both. Why this is,

Fig. 3.

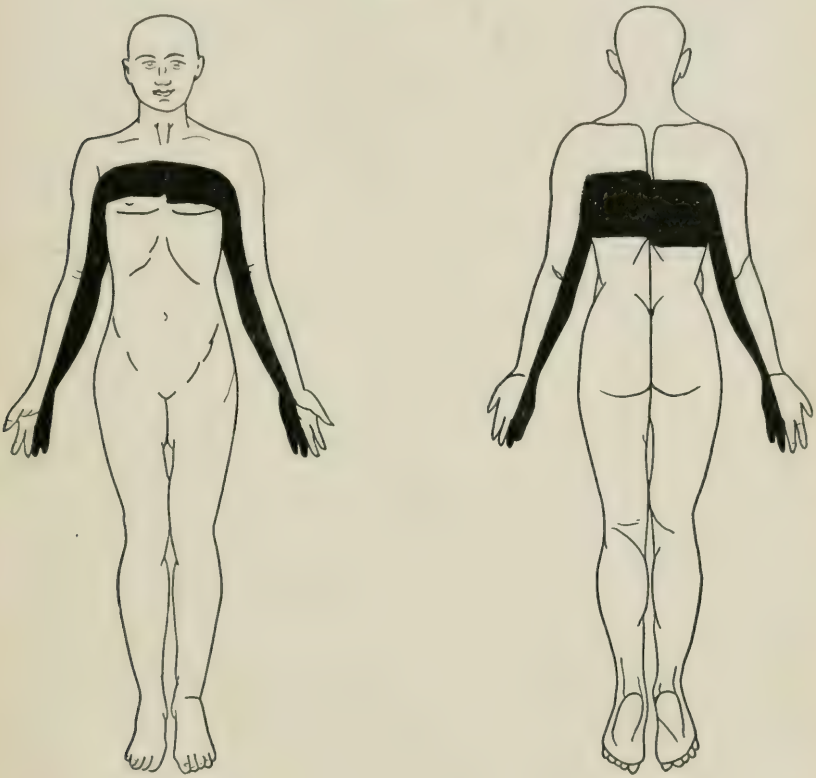


I do not know; possibly a kind and beneficent Providence has decreed that either is enough.

10. *Trunk Anæsthesia.*—This is a symptom of tabes which has been only recently exploited, although, of course, it was always present. Hitzig called attention to it about ten years ago, and then Lachr, a German, examined a number of cases and found the symptom, after which came the communication of Patrick, and now it

is a recognized symptom of locomotor ataxia. Why it was not discovered before, I cannot imagine. Generally there is a diminution of tactile sense, not of pain sense, in the form of a band about the body at about the level of the nipples, as shown in Figs. 2 and 3. In this patient we can demonstrate a narrow band of tactile anæsthesia. There are exceptions to the rule that the area is anæsthetic

FIG. 4.



and not analgesic. Sometimes it is blunted to painful impressions as well as to touch. When the anæsthetic zone reaches as high as the second rib, it is likely to extend on to the arms, as shown in Fig. 4. This symptom is present in about eighty per cent. of well-defined cases; taking the incipient cases only, it would probably not be present in more than forty per cent., still that is a good many. Figs. 2 and 3 illustrate the condition.

11. *Ulnar Analgesia.*—By this is meant loss of the pain which is normally felt when the ulnar nerve is forcibly pressed against the inner condyle of the humerus. Aside from the tingling in the arm and in the fingers felt when the ulnar nerve is struck or forcibly pressed upon, there is a distinct sensation of pain at the point of pressure. When, in this patient, I press the ulnar nerve very forcibly against the inner condyle of the humerus, you notice that, although there is a distinct twitching of the fingers and some of the forearm muscles from the force of the pressure, he evinces no discomfort whatever. Sometimes the symptom is present on only one side. The patient who is blind has no ulnar analgesia, no trunk anæsthesia, and no incoördination.

12. *Testicular Analgesia.*—By this is meant absence of the peculiar disagreeable and painful sensation produced in the normal man by compression of the testicle. Pitres asserts that this symptom is present in seventy-five per cent. of male tabetics.

13. The various *crises* of which I have spoken.

14. *Incoördination.*—I mean incoördination discovered by examination. To wait for the full-fledged, typical, wide-footed, ataxic, heel gait is to wait for the whole clinical house to fall on us. It is proper to add that possibly some neurologists would place this symptom somewhat higher in the diagnostic sequence.

15. *Diminution of Sexual Power.*—Very frequently associated with some anæsthesia of the penis, especially of the glans.

16. *Hypotonus.*—This is a diminution of the natural muscle tonus. It is not of very great diagnostic importance, but it is always present in the more advanced cases and sometimes in the incipient ones. The muscles lose their ordinary tone and their natural resistance to passive movements. You can double up such a patient like a jack-knife, and it causes him no pain, and but little force is required. Fig. 5 shows a patient thus doubled up as in the treatment to be mentioned presently; and although this patient has no symptoms whatever referable to the trunk or legs, and although this was the first time he had been flexed, yet the muscular hypotonus was such as to allow of very considerable flexion with slight force and no discomfort whatever.

17. Perhaps more important than the preceding is *impaired muscular sense*, or, as I prefer to call it, impaired sense of motion or position. With his eyes closed the patient is unable to perceive

slight passive movements made by the examiner. This symptom may often be elicited when ataxia has not yet appeared.

18. *Persistence of Painful Impressions.*—A momentary painful stimulus, like a deep pin-prick or severe pinch, is perceived as a prolonged stinging or burning.

19. *Delayed Conduction of Sensory Impressions.*—With reference to the prognosis, I shall make only three statements. First. Some cases are exceedingly slow; the disease continues for years, —fifteen or twenty,—or may come to a stand-still. Second. There are very rapid cases which lead to complete disability within a year. I have seen a number of such. Third. Although no physician can cure locomotor ataxia, there is scarcely a symptom that cannot be relieved in some degree,—at least for a time. This statement seems inconsistent, but it is not. Although the cases ultimately, as a rule, grow worse, yet the experience of every neurologist, as well as my own, is that we can nearly always relieve these patients somewhat if they are in a position to take the treatment prescribed.

Regarding the therapy, there are only two or three points that time will allow me to mention to-day. The first is this: In many cases there is added to the natural physical disability of the disease a functional disability because of the disease. What I mean by that is that a patient having a certain amount of inevitable disability, be it failure of vision, double vision, incoördination, lightning-like pains in the legs, gastric crises, or the prostration which accompanies such crises, becomes nervous. For instance, he is worried about himself because he cannot walk well, and becomes timid and apprehensive. He is afraid to cross a street; upon his incoördination is developed typical agoraphobia. In short, he acquires such a fear of crossing a street that, although physically able, he is functionally unable to try. Another patient with lightning pains gets all sorts of depressing ideas about himself. He may become neurasthenic or hysterical. The patient with gastric crises nearly always has some explanation for his vomiting spells, or, as he is likely to call them, bilious attacks, and the physician is only too prone to fall in with his opinion or formulate an erroneous one of his own. Between them, then, they eliminate one aliment after another until, in the course of a few years, there is scarcely a single article of diet that the patient feels he can eat with impunity, and he becomes a

confirmed gastric hypochondriac. I might thus go through a long category of functional troubles which such patients have, but which do not necessarily belong to locomotor ataxia, and which readily yield to rational treatment.

The best treatment for the incoördination is that of re-education by means of systematic movements or exercises. This system of exercises was developed in this way: A certain patient with locomotor ataxia could not touch the tip of his nose with his eyes closed at the first examination that was made by his physician, Dr. Frenkel, of Switzerland. When he returned a week later, it was found that he could execute this manœuvre with ease and accuracy, and he stated that he had been diligently practising the movement every day. This set the Swiss physician to thinking. He reasoned that if a patient with tabes could do this, he could do something more, and thus he evolved an elaborate system of exercises for the treatment of locomotor ataxia. As developed by Fränkel, the system requires quite a lot of apparatus, but the principle once grasped, special appliances are quite unnecessary. Every patient has at home cracks in the floor or stripes on the carpet that he can walk, "heel and toe;" every patient can make spots on the floor at different distances and in different figures, and practise touching these in rhythmic rotation with one foot, while standing on the other; every patient can sit in a chair and pass the foot first over and then under a cane resting on two chairs in front of him. The idea is simply to practise uncomplicated movements with accuracy and freedom of motion, and but little ingenuity is required to devise a variety of such calisthenics.

Of late years few remedies have been more abused in the treatment of nervous affections than electricity. If I were to give a course of lectures on electro-therapy in nervous diseases, I should give about three lectures explaining its uses, and about seven lectures explaining the cases in which it should not be employed, because it would either be of no use or it would do harm. There is a host of nervous affections for which electricity is used in a haphazard, happy-go-lucky fashion without any results whatever from the electricity itself. There are a few affections in which electricity, when properly used, is of distinct advantage. One of these I believe to be tabes. Electricity, however, has never cured a case of tabes, but I think it helps materially. It is particularly good for the

bladder disturbance, for the lightning pains, and for the diminished sexual power. To use electricity for this purpose we must have a strong galvanic battery and very large electrodes; a faradic battery will not accomplish much, although occasionally a strong faradic current is useful for relieving the lightning pains.

In very many cases of locomotor ataxia there is residual urine, and if this be drawn once or twice in twenty-four hours the patient does not have the vesical trouble, which is sometimes exceedingly annoying. Not long ago a tabetic was sent to me whose only complaint was that he wet his trousers every day; that he had to urinate every hour during the day; and that, in spite of all precautions he took, he would soak an enormous diaper at night. By simply drawing this man's urine every morning and night (under careful instructions he was able to do it himself), he was in a large measure relieved of both diurnal and nocturnal distress.

Suspension helps a good many cases; especially the lightning pains, vesical trouble, and incoördination are apt to be relieved by this means. There is a substitute for suspension which seems to be equally good,—that is, flexion of the patient, as I now show you with Case III. He lies on the back and brings the feet up over the face, where I grasp them and exert considerable pressure downward. This pressure is maintained for from two to five minutes, and the treatment may be administered once daily. Ordinarily some member of the family may attend to it after having been instructed by the physician (Fig. 5).

To relieve the lightning pains, aluminum chloride in doses of from three to five grains, t. i. d., has been recommended. I cannot tell you whether it is of much advantage or not, because I have always used it in conjunction with other remedies. It certainly has not benefited some cases; in others it seems to do considerable good. It has been recommended by Sir William Gowers, than whom there is no better neurologist and observer; consequently it is worthy of trial. Hypodermic injections of atropine, given in large doses, often relieve these pains. Morphine should be employed as a last resort. Do not use it if you can get along without it.

ACROMEGALY.

CLINICAL LECTURE DELIVERED AT THE MASSACHUSETTS GENERAL HOSPITAL.

BY G. L. WALTON, M.D.,

Clinical Instructor in Neurology in Harvard University; Physician to the Neurological Department of the Massachusetts General Hospital.

GENTLEMEN,—The patient I show you to-day is forty-four years of age, and is, as you see, in some respects a very large man, though less than six feet in height. His chest is deep, his features large, his lower jaw thrust forward; his hands and feet are enormous. The measurement of the hand from the carpal border of the palm to the tip of the middle finger is eight and three-quarters inches; the width is four and one-half inches. The feet are proportionately large, measuring twelve and one-quarter inches in length. The head is large, though not in proportion to the hands and feet. We find the cranial circumference to be twenty-six inches, and the circumference of the chest forty-six inches. These dimensions are among the largest recorded, though they have been exceeded. In two instances the feet measured over fourteen inches.

The symptoms of which the patient complains are muscular weakness, pains in various parts of the body, and vague sensations of numbness and prickling without real inability to feel objects, —namely, subjective sensory disturbances, which we may designate as paræsthesiæ.

The diagnosis is so obvious in this case that we may proceed at once to the symptoms commonly found in acromegaly. These may be divided into three classes: 1. Symptoms of disordered metabolism,—namely, excessive growth of osseous and connective-tissue structures. 2. Symptoms of pressure, such as cranial nerve paralysis, pyramidal tract paralysis, and diabetes mellitus; to this category belong optic atrophy from pressure of the nerve against the orbital plate, and, preceding the atrophy, various disturbances of the visual field resulting from pressure upon the optic commissure

FIG. 1.—Dr. Walton's case of acromegaly.

FIG. 2.—Portion of skiagraph of left hand of Dr. Walton's case of acromegaly. (Actual size.)

either anteriorly or posteriorly, the most common finding in this connection being a loss of vision for objects on the temporal side of the field. Hemianopsia appears less frequently, and is due to pressure on the optic tract behind the chiasm. 3. General symptoms,—headache, somnolence, and lack of mental vigor, whether resulting from pressure or from the disorder of pituitary secretion. Under which of these heads the paræsthesiæ and pain should be included it is perhaps difficult to determine, but it seems not unreasonable to attribute these symptoms to irritation and pressure caused by increase of connective tissue about the nerve-fibres.

We find, on inquiry, that while the patient's hands and feet have always been large, their size has increased greatly within the last five years; we find also that the projection of the lower jaw has steadily increased during the same time, previous to which the upper and lower teeth met evenly, whereas the lower teeth are now projected forward a half-inch. This establishes the fact that the affection is a steadily increasing disorder of nutrition, and not a mere natural anomaly. As to the symptoms of local pressure, we obtain negative answers regarding double vision, impairment of eyesight (other than that naturally accompanying advancing years), disorders of olfactory sense, or other cranial nerve-function. There is no complaint of localized loss of power. With regard to general symptoms of brain tumor, we find there is no headache, vomiting, somnolence, or loss of memory. The physical examination confirms the statement made with regard to paralysis; we find no spastic gait, no local paralysis, no disturbance of reflexes or of objective sensation. We find vision good, the fundus normal, the visual field complete. In answer to your question whether the bones participate in the hypertrophy, I need only show this excellent skiagraph. You will note that the bones are proportionately enormous. You will observe also the comparatively hazy outline and open structure of the ungual phalanges in comparison with those of the healthy hand, a phenomenon which is noted as characteristic of acromegaly. The abnormal projections at various points, for example, on the ulnar side of the proximal end of the first phalanx of the index-finger, doubtless represent local hyperplasia at the points of insertion of tendons and fasciæ. (Figs. 1 and 2.)

Disease of the pituitary body has been found in too large a proportion of cases of acromegaly to be explained by mere coinci-

dence; their interdependence is so generally recognized that we may accept it without further discussion. This fact being accepted, it is easy to explain the occasional appearance of pituitary tumor without acromegaly when we remember that a very small portion of a ductless gland is capable of carrying on its duties; and if we accept the theory that acromegaly is produced by increased secretion, it is easy to see that a tumor may exist without producing this result. The absence of obvious tumor, again, does not preclude disorder of function. The question of the pathogeny is narrowed, therefore, to a study of the relation of the glandular disorder to the symptoms.

Much discussion has taken place as to whether the pituitary tumor should be regarded as the essential etiological factor, or as one of the results of the disordered metabolism, such disorders being caused by some more general condition classed under the general head "trophoneurosis." Without entering into this discussion, it will suffice to state that the former view,—namely, that of primary pituitary gland disease,—seems most worthy of credence. If we accept this view, the only question left is whether nutrition is affected by deficiency or excess of glandular secretion? Is the secretion analogous to that of the thyroid gland, and does it destroy toxines or in some other way check hyperplasia of tissue; or is the secretion, on the other hand, itself an irritant, and does its excessive production act as a stimulus to growth? The arguments in favor of the latter supposition seem most convincing,—namely, (1) post-mortem examination has revealed complete destruction of the pituitary gland without symptoms of acromegaly; (2) the removal of the pituitary body in lower animals has given rise to no symptoms analogous to those of acromegaly; (3) feeding with pituitary extract has not had the least influence upon the progress of the disease, in marked contrast to the results of thyroid feeding in myxœdema.

It may be urged in opposition to this theory that sarcoma rather than adenoma, or gland hypertrophy, predominates in the post-mortem reports. In this connection, however, it is of great interest to note that Brooks, whose systematic and painstaking investigations render him particularly worthy of quotation, reported his own first cases as sarcomata; but further study led him to observe the great similarity of the cells in a hypertrophied pituitary gland to

those of sarcoma, and after convincing himself that his later cases were adenomata, he examined again his earlier cases and changed the diagnosis. In view of this fact, we are certainly justified in allowing the possibility that individual post-mortem reports may be misleading.

I will submit for your examination several slides prepared from an undoubted angiosarcoma of the pituitary body. You will note, in the first place, rounded masses, light in color, and of homogeneous appearance; these are collections of colloid, the pituitary secretion. One of these masses is a quarter of an inch in diameter. The function of the gland was probably, therefore, increased rather than diminished in activity up to the last, even though the growth is a sarcoma, not an adenoma. The rapid progress of this particular case after the appearance of cerebral symptoms certainly coincided with the history of sarcoma, this history being in marked contrast with that of typical acromegaly, which may extend over a period of twenty years without the development of serious symptoms, and with no sign of metastasis.

I will remind you that the pituitary body consists of two parts entirely distinct in form and function, and separated by a fibrous lamina. The hypophyseal portion is a rounded mass, resting in the sella turcica, continuous with the infundibulum, a mere outgrowth of brain-tissue, subserving no known function; anterior to this mass and separated from it by a fibrous septum lies the prehypophysis, presenting a concave posterior surface. The prehypophysis constitutes the only glandular portion; it has no connection with either the brain or the hypophysis proper, but is developed from the ectoderm of the buccal cavity. In certain lower vertebrates a so-called water vascular tube serves as a duct, but in the more highly organized types this tube has disappeared, and the human pituitary gland belongs to the class known as ductless, its secretion being taken up by the lymphatics. It is only when the glandular structure is diseased that symptoms of acromegaly appear; it has been claimed that this is always the portion first invaded in case of pituitary new growth.

If hyperplasia affects the pituitary gland in infancy, the result is not acromegaly, but gigantism, the long bones growing rapidly longer till a height perhaps of seven and a half feet is reached. I pass round illustrations of the skeleton of the so-called "Amer-

ican Giant," carefully studied by Hinsdale. You will note various points of similarity between this condition and acromegaly: the type of face is the same,—namely, long and asymmetrical, with particularly long lower jaw thrust well forward. The thorax in both is very deep in proportion to its circumference, and the hands and feet are long and large in both affections. In acromegaly, kyphosis is apt to add to the thoracic deformity. Giants, like acromegalics, as Hinsdale points out, far from possessing the strength their size would suggest, are generally weak, at least after the disease is well established.

It has been already noted that peculiarities of form or feature suggestive of acromegaly have in certain cases preceded the onset of serious symptoms; this fact renders it not improbable that congenital abnormality of the pituitary gland may furnish the basis for later neoplasm. In pursuance of this line of thought, it would seem pertinent to inquire whether variations in the secretion of this gland within physiological limits may not determine the character of growth in the individual and in the family, and whether among healthy individuals the tall stature, the long face, the prominent jaw, the deep chest, and the large hands and feet may not represent a liberal secretion of the normal pituitary gland.

Surgery

THE MODERN OPERATIONS FOR THE RADICAL CURE OF INGUINAL HERNIA.

BY EDMUND ANDREWS, M.D., LL.D.,
Professor of Clinical Surgery in the Northwestern University, Chicago.

THE plan of operating by free incision for the radical cure of hernia has triumphed over all others. This victory was made possible by the evolution of antiseptic surgery thirty years ago. Among the tentative methods previously employed, all of which were either dangerous or ineffectual, may be mentioned the following:

Cure by Pressure.—Hood's trusses were invented to induce "adhesive inflammation," and thus close the hernial canal. These failed in nearly all cases.

Scarification and Ligature of the Sac.—The inner surface of the hernial sac was scarified by ingenious instruments especially devised for this purpose, the sac itself being sometimes ligated outside the external ring. This procedure also failed.

Invagination of the Scrotum.—Folds of the scrotum were tucked up into the inguinal canal and held there by stitches for eight days. The folds, however, afterwards retracted and left the channel still pervious. A modification of this operation by Wützer, of Berlin, in 1838, enjoyed a temporary popularity, but it also proved unsatisfactory.

Subcutaneous Sutures.—After the introduction of antiseptic methods by Lister, John Wood, of London, ventured on a slightly bolder operation. With due antiseptic precautions he introduced wire into the external ring, and by curved needles set in handles contrived to sew the walls of the inguinal canal together. The wire was removed in about ten days. Far better results were thus ob-

tained than by any previous method, but this operation has fallen into disuse.

Injection Methods.—About 1820 Velpeau, of Paris, and Pancoast, of Philadelphia, simultaneously began the hypodermic injection of iodine into the hernial canal, hoping by this means to effect its obliteration on the principle that had proved so successful in hydrocele. Pancoast reported several cases. A student from Massachusetts, named Heaton, who had attended Pancoast's clinic, conceived the idea that a watery extract of white-oak bark would be more astringent, or, in his Yankee dialect, "more puckering," than iodine, and therefore more likely to cause contraction and obliteration of the canal. He became an advertising itinerant, and traversed all New England, injecting his secret extract and gathering in his fees. His idea got out, and became the stock in trade of a multitude of travelling quacks all over the United States. This plan was tested by many surgeons, but it was found that in the great majority of cases a temporary cure was followed by relapse.

In 1870 Schwalbe, of Germany, injected alcohol varying in strength from twenty-five to seventy per cent. into the connective tissue about the sac. The process was very painful and slow, and fell into disuse.

Lannelongue and Demars, of France, injected a ten per cent. solution of zinc chloride, and claimed immense success, but their method did not become permanently established.

OPERATIONS BY INCISION.

After the value of asepsis and antisepsis had become fully established, surgeons began to attack hernia through open incisions, and were thus able to repair the damaged parts while having every step of the operation clearly under their eyes.

Over one hundred varieties of open operations have been published, many of which are virtually identical, but they can all be classed in a few groups.

The Bassini Group.—As early as 1870 Professor O. Marcy, of Boston, operated by an open incision, and a year afterwards published his cases in the *Boston Medical Journal.* Several years later, Professor Bassini, of the University of Padua, Italy, independently developed a similar operation. Before printing anything he spent a number of years in perfecting his technique and in observing the

permanence of the cures. Finally, in 1886, and again in 1890, he published his cases. Bassini's papers attracted world-wide attention and admiration, and by general consent his operation and numerous similar ones devised by others were called after his name without much regard to minor differences or to priority of invention.

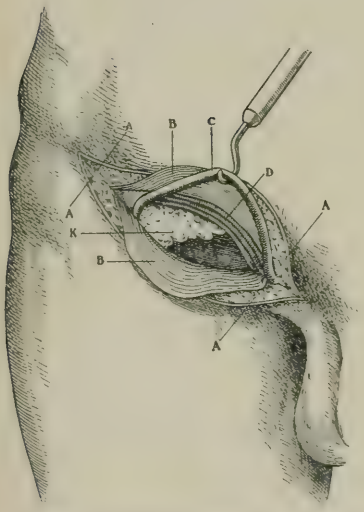
In order to give his technique in its very latest form, I have condensed a description of it from manuscript notes taken by Professor E. Wyllys Andrews, of Chicago, at Bassini's clinic a few weeks ago. The same writer has a fuller article on the subject in the *Medical Record,* October 28, 1899.

Bassini is very precise and unvarying in all his steps, so that his assistants have learned exactly what to do, and everything proceeds rapidly, the average duration of the operation being about twenty-two minutes. All the usual aseptic and antiseptic precautions are strictly observed.

The skin and subcutaneous adipose tissue over the inguinal canal are lifted up into a fold about two and one-half inches high, crossing the direction of the inguinal canal at right angles. A stroke of the knife parallel to the canal cuts this fold down to the external oblique muscle. A few more touches divide the fibres of the external oblique muscle in a direction parallel to their course and expose the canal. The lower flap of the external oblique fascia is then raised or dissected up by the handle of the scalpel as far as Poupart's ligament. Lobules of fat in the canal are stripped away, and the hernial sac near the neck is separated from the other tissues and from the cord. The finger is now inserted alongside the sac into the internal ring, and detaches the peritoneum from the abdominal wall for a distance of two centimetres all around the inside of the ring. The sac is now opened at the lower part and examined. The contents are reduced, portions of omentum being ligated and cut off if necessary. A clamp is placed high up on the neck of the sac, and the sac itself is ligated above the clamp. If the sac is large, the ends of the ligature are passed through it before tying. The sac is now cut off below the clamp, the clamp itself is removed, and the stump drops back into the abdomen. Bassini retracts the edges of the external oblique fascia and lifts the cord out of the wound, retaining it with a forceps and a loop of silk or a blunt hook. Stitches are next in order. Having lifted the ring and adjacent

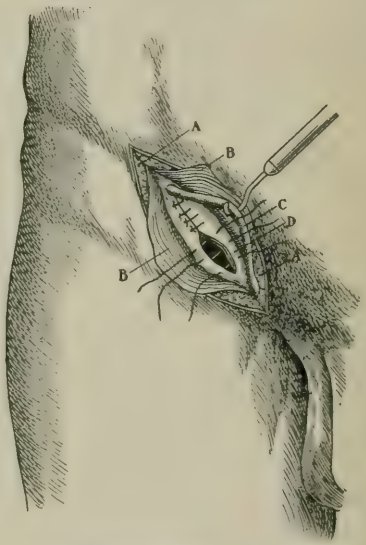
parts forward by inserting a forceps and a flat director, the needle may be easily passed without wounding the peritoneum. The first or deep line of stitches begins near the pubis. The first one or two stitches may have to enter the rectus muscle. The needle pierces the transversalis and internal oblique a little over a centimetre above their lower border, and passing down comes out at their margin. It is then carried down across the wound behind the cord, and thence

Fig. 1.



Bassini's operation, showing the wound before suturing: A, A, A, subcutaneous fat; B, B, reflected flaps of external oblique aponeurosis; C, spermatic cord lifted out of wound; D, deeper layers of abdominal walls; K, extra-peritoneal fat. (After Bassini, in *Archiv f. klin. Chir.* vol. xl. p. 429, 1890.)

Fig. 2.

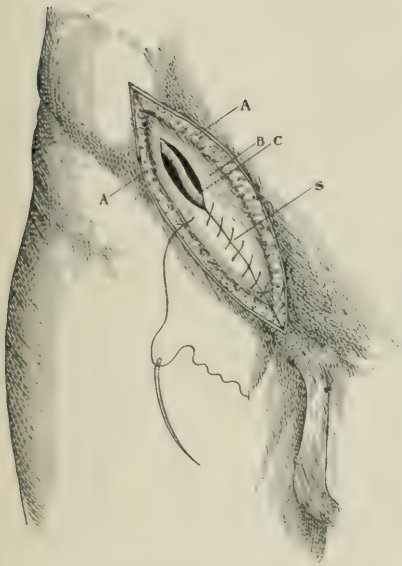


A, A, subcutaneous fat; B, B, reflected external oblique aponeurosis; C, cord lifted out of wound; D, deeper layers of abdominal wall being sutured behind the cord. (After Bassini, *Ibid.*)

passed from within outward through the reflected lower edge of Poupart's ligament, carefully avoiding the great vessels. From four to six of these stitches are inserted before any of them are tied. They are generally somewhat over a centimetre apart. Bassini begins to tie them at the upper end of the row. He thus repairs the internal ring and constructs a solid floor for the canal. The cord is now laid into the wound, and the aponeurosis of the external oblique is drawn together over it and closed with a continuous silk

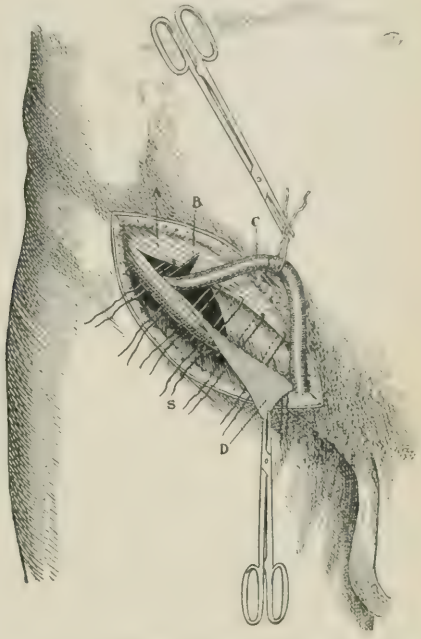
suture. Finally, the integuments are brought together by a continuous button-hole suture. The silk used is previously boiled in glycerin. The wound is dressed with corrosive sublimate gauze, which is not changed for eight days. The patient is kept very quiet in bed for some days by a light starched bandage, and afterwards

FIG. 3.



A, A, subcutaneous fat; B, external oblique aponeurosis being closed over the cord; C, spermatic cord; S, continuous silk suture partly completed closing the external oblique aponeurosis over the cord. (After Bassini, *Ibid.*)

FIG. 4.



E. W. Andrews's imbrication or lap-joint operation, showing the deepest line of stitches inserted but not yet tightened: A, upper flap of external oblique aponeurosis; B, notch cut in the same to receive the cord; C, the cord lifted from its bed; D, lower flap of external oblique aponeurosis reflected downward; S, line of mattress stitches for drawing the upper flaps of the external oblique, the internal oblique, and transversalis down behind the cord to Poupart's ligament.

goes about his business without truss or bandage and without any restrictions as to his customary exercises. The relapses are less than five per cent.

The Lap-Joint, or Imbrication, Method of E. Wyllys Andrews, of Chicago.—This modification of Bassini's operation differs from it in that the upper segment or flap of the external oblique aponeu-

rosis is drawn down behind the spermatic cord, while the lower flap is drawn up in front of it, the two flaps thus lapping, or imbricating, the cord being included between them. The steps are as follows: After opening the integuments the aponeurosis of the external oblique is split parallel to its fibres the whole length of the inguinal canal. The hernial sac is opened and its contents reduced; it is then ligated as high up as possible. The finger is passed into the internal ring and gently separates the peritoneum from the inner surface of the abdominal wall around the ring for a distance of about two centimetres from its circumference. The sac is now pulled down, cut off below the ligature, and removed. The spermatic cord is next stripped of lobules of fat, lifted temporarily from its bed, and held aside by retractors. The internal ring is repaired, and the posterior wall of the canal reconstructed by drawing down all the muscles and fasciæ of the upper flap behind the cord and stitching them to Poupart's ligament below by mattress stitches in the following manner:

1. A needle with a suture is passed from without inward through Poupart's ligament, and then upward across the canal, and there picks up the upper flap passing from within outward through the transversalis fascia, the internal oblique muscle, or conjoined tendon, and emerges through the external oblique aponeurosis, thus embracing all the layers of the abdominal wall.

2. The needle now returns across the wound parallel to its former course, and emerges again from Poupart's ligament not far from its point of original entrance, thus making a "staple," or mattress, stitch. From three to six such stitches complete the suture line.

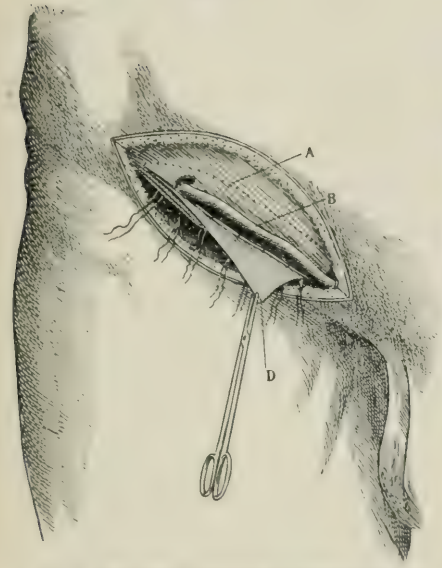
3. The upper two stitches are tied first, and the pulse of the cord felt before and after tightening them, to make sure the vessels are not constricted. The remaining stitches are now tied; the knots will all be outside the ligament, and a solid floor is made for the inguinal canal.

4. The spermatic cord is now laid on this floor and the lower flap of the external oblique is drawn up over it and stitched in the line indicated by the dots shown in Fig. 5. By this imbrication, or overlap, the cord is provided with a new inguinal canal, having a very firm wall behind it. The skin is closed with a buried suture and the wound sealed without drainage.

Over five hundred operations by this method are reported by ten or fifteen different operators, without any deaths, and with less than five per cent. of relapses.

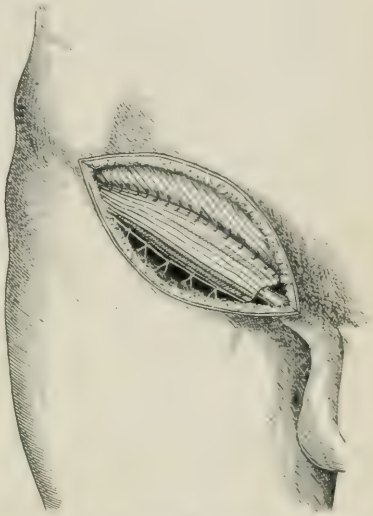
Method of Halsted, of Baltimore.—This also belongs to the Bassini group, but was published a little earlier than the method of the Italian surgeon. The hernial sac and the internal ring are laid open in very much the same way as in Bassini's operation. An

FIG. 5.



E. W. Andrews's lap-joint operation, showing lower flap of the external oblique ready to be drawn up over the cord and lapped, or imbricated, upon the upper flap: A, the dotted line on the upper flap of the external oblique shows the place to which the lower flap is to be drawn up and sutured; B, spermatic cord.

FIG. 6.

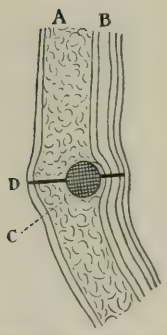


E. W. Andrews's operation, showing the lower flap of the external oblique lapped over the cord and sutured to the front of the upper flap above it. The cord is seen emerging from the new canal between the two layers of the external oblique.

incision is then made from the outer border of the internal ring extending upward and outward for three centimetres into the lower border of the internal oblique muscle. The sac is opened and its contents reduced, some omentum being amputated if necessary. The neck of the sac is stitched together with fine mattress stitches, the sac cut away just below the seam and removed. The cord is now lifted out, and enlarged veins are sometimes removed. The cord itself is made to emerge from the notch cut in the lower border of the in-

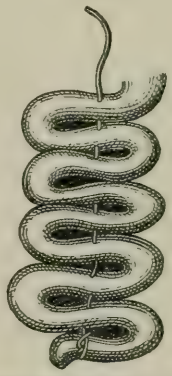
ternal oblique muscle, thus passing through a new ring. The cord being held up, the upper flap of the external oblique, internal oblique, and fascia transversalis are all drawn down behind it and stitched to the corresponding tissues of the lower flap. The cord now lies on the external surface of the external oblique fascia. The skin and subcutaneous adipose tissue are closed over it in the usual manner. Halsted takes pains that the cord shall not be constricted at its point of emergence from the notch cut in the internal oblique muscle; and if veins have been removed he does not use the notch, lest the circulation of the testicle be interfered with. Halsted prefers silver wire for sutures. His operation has secured excellent results. A full description of the technique is given in the *Johns Hopkins Hospital Reports,* vol. vii.

FIG. 7.



Showing the position of the cord in Halsted's operation: A, skin and superficial fat; B, deeper layers of the abdominal walls; C, position of the spermatic cord; D, incision closed in front of and behind the cord.

FIG. 8.



Macewen's method of folding the sac to form a plug for the internal ring. (After Treves's *Operative Surgery*, Lea Bros. & Co., Philadelphia.)

FIG. 9.

The folded sac placed behind the internal ring in Macewen's operation. (*Ibid.*)

CLASS OF OPERATIONS IN WHICH THE SAC IS USED TO CLOSE, OR PLUG, THE INTERNAL RING.

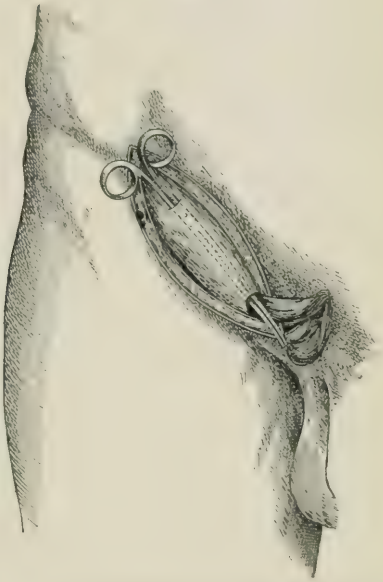
Macewen, of Glasgow, devised the best known method of this group. Opening the integument by suitable incisions, he exposes the hernial canal by dividing the external oblique parallel to its fibres. The sac is then separated from the cord, opened, and its contents reduced. By a finger inserted through the internal ring alongside the sac, the peritoneum is separated from the abdominal

wall for half an inch all around the ring. A thread is now firmly stitched to the lower end of the sac with a needle made to traverse it to and fro through its whole length. The needle is then passed into the internal ring, and finally brought out through the muscles and the fascia of the external oblique an inch to the outside of and above the internal ring. By pulling on the thread the sac is drawn up like a curtain into a folded bunch, which slips through to the inside of the ring, plugging it on the inner side. The thread is

FIG. 10.

Macewen's operation, showing the method of stitching the lower border of the conjoined tendon to Poupart's ligament. (After Treves's *Operative Surgery*, Lea Bros. & Co., Philadelphia.)

FIG. 11.



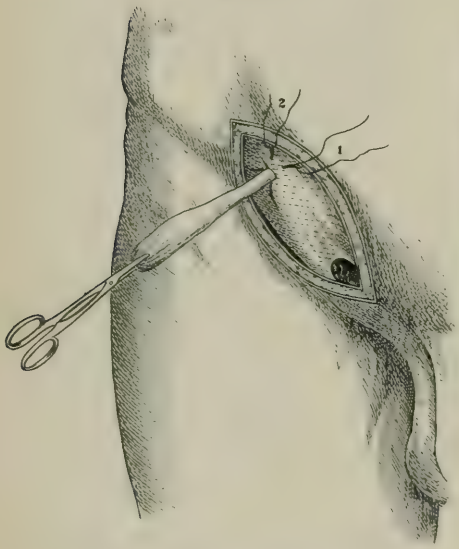
Kocher's operation, showing the manner of seizing the bottom of the sac in order to draw it up through the inguinal canal. (Kocher's *Operative Surgery*.)

fastened above to the muscles, to keep the plug in place. The conjoined tendon and other tissues are drawn down and stitched to Poupart's ligament, covering in the cord. The integument is closed in the usual way. Macewen's operation is very ingenious, and attracted much attention, but is not as satisfactory as Bassini's.

Barker's Operation.—This resembles Macewen's in the use of the folded sac to plug the internal ring, but differs from it in leaving the scrotal portion undisturbed.

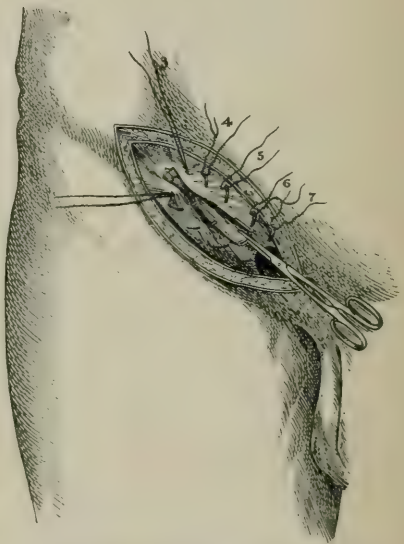
Th. Kocher, of Berne, Switzerland, devised another method of utilizing the hernial sac. Having laid bare the obliquus externus, he opens to the sac just below the external ring, separates the sac and draws it up out of the scrotum. A small opening is then made through the muscles at a point a little above and to the outside of the internal ring. A long forceps is passed into this opening and thence along the inguinal canal outside of the sac until it emerges from the external ring. The inguinal canal is not opened in front.

FIG. 12.



Kocher's operation, showing the sac well pulled out through the abdominal wall. (Kocher's *Operative Surgery.*)

FIG. 13.



Kocher's operation, showing the sac folded down and stitched in front of the external oblique. (Kocher's *Operative Surgery.*)

The fundus of the sac is seized with the forceps, and drawn up the whole length of the canal and out through the opening made in the muscles above, where it is fastened with stitches. The sac is then either cut away outside the point at which it is attached, or is placed parallel to the fibres of the external oblique fascia, where the fascia is folded over it and stitched to it. The integuments are closed as usual.

The ingenuity of the technique and Kocher's great name as an operator have drawn much attention to this operation, but it is in-

ferior to several of the Bassini group. C. B. Ball, of England, twisted the sac very tight and stitched its firmly contorted stump inside the internal ring, so as to block up the opening. The value of his method is not yet known.

BRIEF MENTION OF MISCELLANEOUS METHODS.

Kelly and several others have brought down thick flaps from the rectus abdominalis muscle in order to strengthen weak or deficient posterior walls of the canal.

Wölfler used for this purpose a flap from the anterior sheath of the rectus. Others have turned up flaps from the strong fascia lata of the thigh or from the tensor vaginæ femoris muscle, and some early surgeons stitched the testicle into the inguinal canal. Others have inserted bone-grafts from distant parts of the body, and some have tried sponge-grafts.

Czerny and Fowler have used crossed, or "shoe-laced," sutures in drawing the ring together. Fowler also places the cord inside the deep abdominal muscles.

McBurney tried to close the wound by granulation, so as to block the channel with a thick mass of tough cicatricial tissue. This plan was regarded by the profession as offering a strong probability of success, and was received with much favor; but the cicatricial tissue slowly yielded, and there were many relapses. McBurney himself published this result, and abandoned the method.

Banks's Operation.—Usually Banks does not slit up the inguinal canal, but pushes the finger up through the external ring, loosening the sac from the walls of the canal well up to the internal ring. The sac is then drawn down, ligated as high up as possible, cut off below the ligature, and removed. The rings and the canal are then narrowed by sewing the conjoined tendon down to or near Poupart's ligament. A horsehair drain is inserted and the external wound closed as usual.

Lucas-Championnière, of France, tried a modified cicatricial operation. He closed the skin firmly, but left the deeper parts loose, so as to fill the whole canal with a "voluminous mass of scar tissue" and block the channel. But as the parts are flexible, the walls of the canal must fall together and be held in contact by the dressings. It is therefore doubtful whether the supposed interior mass of scar tissue is as "voluminous" as he imagines. If it were, it would

probably stretch and give way, like the still more massive cicatrices secured by the McBurney open treatment, which had to be abandoned on account of the yielding of the new tissue. Lucas-Championnière's results are said to be good, probably because the "massive" scar which he seeks for does not actually form.

Frank, of Vienna, cuts a deep groove in the upper border of the pubic bone, lays the spermatic cord in the bottom of the groove, and draws the periosteum over it.

Many efforts have been made to collect statistics in order to ascertain the relative value of the principal operations. After much study of them I regret to say that they have not yet furnished a basis for decisive comparison between the different methods. I have therefore not introduced statistical tables into this essay. When a multitude of enthusiastic surgeons are trying to tabulate the results of their new operations, enormous blunders always occur, as every trained statistician very well knows. Nevertheless, we are indebted to the operators for their work, and for putting valuable facts on record. We shall in time attain correct statistics; meanwhile we can safely draw the following general conclusions:

1. The superiority of treatment through open incisions seems to be firmly established.

2. The great number of new operations included in the Bassini group shows that the drift of surgical preference is in that direction.

3. The use of the folded sac to block the hernial canal is still strongly urged by a few eminent surgeons, such as Macewen, Kocher, and Barker.

4. All operations of every group make free use of adjacent muscles and fasciæ by transplanting, or rather drawing them in to repair the deficient portions of the abdominal walls.

PRACTICAL RESULTS.

In careful hands, with due aseptic and antiseptic precautions, the principal radical cure operations are almost absolutely free from danger, and the chances for a relapse afterwards are less than five per cent. It must be understood, however, that, as in general surgery, patients laboring under diabetes and albuminuria are not proper subjects for surgical intervention; and as children under

one or perhaps two years of age are usually curable by simple retentive apparatus, they do not require the cutting operation. A few other peculiar classes are also unfit subjects.

With these exceptions, it is perfectly rational to operate on all ordinary cases of hernia. In general, it is safer for the patient to undergo the operation than to run the risks of future attacks of strangulation.

In nearly all cases of strangulated hernia requiring operation the radical cure measures should be superadded to the ordinary technique. It is true that the mortality of these cases is greater than in healthy subjects, but the increased risk is due not to the operation, but to the preceding strangulation.

THIERSCH GRAFTING FOR BURN; RETROVERSION OF THE UTERUS; ABSCESS OF THE LUNG; GENERAL SUPPURATIVE PERITONITIS; DOUBLE INGUINAL HERNIA.

CLINICAL LECTURE DELIVERED AT THE POST-GRADUATE HOSPITAL, NEW YORK CITY.

BY ROBERT T. MORRIS, M.D.,

Visiting Surgeon to the Post-Graduate and Ithaca City Hospitals; Member of the American Medical Association, of the American Academy of Medicine, of the State Medical Society, of the Alumni of Bellevue Hospital, etc.

WITH REMARKS UPON SURGERY OF THE CHEST.

BY JOHN B. MURPHY, M.D.,

Professor of Clinical Surgery in the College of Physicians and Surgeons, Chicago.

THIERSCH GRAFTING FOR BURN.

Case I.—This patient received an extensive burn, and for a whole year there was little attempt made towards repair. The danger of infection in these sluggishly granulating cases is not great, because of local hyperleucocytosis; it is difficult to infect the granulating surface on account of this physiological safeguard,—the leucocytes are there ready for battle. It is this property of hyperleucocytosis which makes many abdominal operations so safe where we have pus to deal with; we need not hesitate to allow pus to flow wherever it will over a normal peritoneum in abdominal abscess cases, knowing that the peritoneum is well protected by leucocytosis. The grayish color of the pus in this patient's case will become sage-colored, due to the presence of the sage-micrococcus, which grows in plaques and has an odor strongly resembling that of musk. This micrococcus often occupies the field at the expense of other bacteria. When this micrococcus occupies the field there are few pathogenic bacteria that seem to make headway. To-day there is not much space left to graft, it being nearly covered by the last grafting. I covered this very large granulating area with Thiersch grafts,

which are now practically all adherent. Remaining patches of granulations will now be covered with blister grafts. They are more fragile than Thiersch grafts, but suitable for small patches. Here I have had prepared a blister, which I cut with a pair of scissors, loosening it all about, and then roll it upon a piece of gutta-percha tissue, which serves as a handle. Now, I have the graft of cuticle already transferred to the gutta-percha tissue. As I unroll it I cut off pieces of sufficient size and place them on the raw surfaces. On account of its being so frail, it is better to apply small rather than large pieces. The overlapping of the graft does no harm; the part that overlaps will disappear.

RETROVERSION OF THE UTERUS.

Case II.—This woman has a retroversion of the uterus, and, as she wishes again to bear children, I shall avoid any of the operations in which the uterus is fastened to the abdominal wall. Alexander's method I frequently use here; but in this operation the adhesions should be separated by opening the peritoneum along the ligaments. If adhesions are not separated, they cause much distress afterwards; and this is one of the reasons why Alexander's original operation is not so good as the one we are about to perform. The ilio-inguinal nerve is liable to be pinched, and causes much discomfort. I like to make a short median incision, bringing the round ligaments through the new opening in the rectus muscle and stitching them together in the middle line, leaving the round ligaments long enough to hypertrophy in pregnancy. This operation of drawing the round ligament through the median opening and fastening it to the pubic region is called Doleris's operation. Having made a short incision in the median line, beginning one and a half inches from the symphysis pubis, the linea alba and the anterior sheath of the rectus muscle are exposed and incised, and the peritoneal cavity is opened. The uterus I find firmly retroverted in the pelvis. I have now the round ligament and Fallopian tube in my fingers, and I am breaking up the adhesions. If Alexander's operation had been tried in this instance, it would not have succeeded well, and would have been followed by much discomfort. I am working by the sense of touch rather than by that of sight. The adhesions are quite firm. I shall next draw the ligament through the rectus muscle; this is done on both sides; then the ligament is fastened. The different

layers are closed separately with chromicized catgut, and this is the only way by which we can get a perfect restoration of the structures divided. Small catgut is used for the peritoneum, the finest, which will be absorbed in three or four days, so making a minimum of disturbance to the peritoneum. Having closed the peritoneum, I take one or two turns through the rectus and pyramidalis muscles. This takes a little more time, but it pays. These structures that I have brought together will be practically united by to-morrow morning. One advantage of this operation is that you work through one incision instead of two. The divided structures are neatly coapted. Here is a dead-space, which will be completely obliterated by blood-clots. These spaces, if aseptic, do little harm beyond leaving the abdominal wall less strong. I use no sutures to bring together the fat, relying upon atmospheric pressure to keep the fat in apposition. Subcuticular sutures I prefer to other suturing, in order to avoid stab cultures of the *Staphylococcus albus.* A few clinics ago, in operating for pyosalpinx, I ruptured one of the pus-tubes and flooded the abdominal cavity with pus; yet the wound healed by primary union. The pus flowed over the normal peritoneum in great quantities, and the reason why infection was avoided was probably because we removed most of the pus promptly with hydrogen dioxide, and then closed the wound without drainage. Dr. Clark, of Johns Hopkins Hospital, brought this treatment to us. There are two things that should be borne in mind here: first, the use of the subcuticular suture to prevent stab cultures of the *Staphylococcus albus;* and secondly, approximation of the fatty walls by atmospheric pressure,—never suture the fat. I now apply aristol powder, bichloride gauze, and cotton; this is to prevent bacteria getting through the dressings.

ABSCESS OF THE LUNG.

Case III.—This case of abscess of the lung began as the result of a pleuritis and pleuropneumonia. There were consolidation over the posterior surface and apex, amphoric breathing over the anterior surface of the right lung, and pus in the bronchi, which the patient expectorated by coughing. The cavity there apparently has not closed, and the case has been in a quiescent state for some months. The pus cavity will not close because the adhesions will not allow a proper contraction of the lung. A few years ago a resection of

the ribs would have been done to allow the lung to contract; but at the present time the method of Dr. Murphy, of Chicago, of compressing the lung with nitrogen gas introduced into the thoracic cavity, offers advantages. This allows the lung to be compressed, forces the pus from the cavity into the bronchi, and permits of secondary adhesions taking place. If you can succeed in this way in causing a collapse of the lung, emptying the pus cavity simply by the pressure of nitrogen gas outside it, it is a much better procedure than any so far attempted. We are fortunate in having the inventor of this new treatment with us to-day, who will address you on this subject.

Dr. John B. Murphy (of Chicago).—Gentlemen, the field of surgery of the chest is now only in its infancy. I believe it has a great future, even greater than abdominal surgery. That may seem an extravagant statement to make, but I believe it, and do not think it is an extravagant statement. The great question now is, Can we make surgery of the chest a practical surgery? Can we remove primary foci of tuberculosis? Can we remove them by operation? Can we remove them when they are small? We know that of all cases in which post-mortems are held, seventy-five per cent. had tuberculosis; we know that twenty-five per cent. had active tuberculosis; that fifty per cent. of these died from tuberculosis; the cured cases were by encapsulation; therefore we know that fifty per cent. of the cases of tuberculosis are cured by nature, and if nature can cure such a large percentage, can we not do something? Abscess of the lung may originate from suppurative tubercular processes, and may finally rupture into a bronchus; it may originate from a pneumonic process, from a septic embolus, etc. Now, what is to be hoped from this method of treatment, and what are its dangers? We can cause a cessation of expansion of the lung by such an injection; if no adhesions are present, we can cause approximation of the walls of the abscess and a uniting of the granulations. Cicatricial contraction is not alone sufficient to cause approximation of the walls; in addition, there is the constant pressure present. The pus may come out through the mouth or be inhaled into the other lung; if the latter happens, we get a septic pneumonia, and both lungs would then be incapacitated, one by a septic pneumonia and the other by the abscess. Lemke, of Chicago, re-

ported four hundred injections with only two accidents, and these accidents were readily avoidable. In cases of abscess of the lung in which the pleural cavity is filled with nitrogen gas, when fluid appears it can be coughed up and no harm result from it; but in one instance an operation was performed, an incision made, and the lung collapsed; as it did so, pus came out of the mouth; the lobe was fastened outside the incision, but the next day a pneumonia developed and the patient died If I had attempted to do what Dr. Morris is to attempt to-day, I could have saved that boy. Twenty or thirty cases of hemiplegia have occurred, produced by gas getting into the circulation; none of these cases was fatal, however. Another accident happened to a man on the table, with the needle inserted; the man coughed, the needle injured blood-vessels, and the patient died from hemorrhage. Both these accidents could have been avoided. If adhesions are present, the abscess cannot collapse; an examination on the following day will show whether adhesions exist or not. Remember, that if there are no adhesions, so that the lung may collapse, there is no necessity to open the chest.

Dr. Morris (continuing).—The abscess in this man opened once in the neck, externally, and is now open by way of the right bronchus. I find that adhesions prevent collapse of the lung when the nitrogen is introduced into the pleural cavity, so I now resect a rib behind the right scapula, and, introducing a scalpel into the lung, come to this large collection of pus nearly three inches from the surface. A rubber drainage-tube is introduced and the cavity flushed with peroxide of hydrogen.

GENERAL SUPPURATIVE PERITONITIS.

Case IV.—This case, which I operated upon last Saturday, will undoubtedly recover; she is doing nicely. The chief principles employed in this case were two,—namely, flushing the pus out of the general abdominal cavity, and flushing the veins with saline solution in such quantities that the emunctories could get the toxines out of the circulation. In other words, get the pus out of the general peritoneal cavity and the toxines out of the circulatory system. We have a good many cases of general suppurative peritonitis here that get well, as many members of the class are aware. If you wash out the general abdominal cavity and distend the veins with saline

solution, a good proportion of the cases gets well; but if you neglect either of these procedures, they are almost certain to die. On the day following operation this patient was not doing so well, and I thought it would perhaps be better to give another transfusion; I did not find it necessary to do so, however, and she is now doing nicely.

The longer a case of general suppurative peritonitis is under way the more leucocytes are manufactured for warring against the bacteria. At the end of a few hours the polynuclears are greatly increased in Nature's attempt to destroy any bacteria that are present. Hyperleucocytosis is at the bottom of these recoveries; at the end of twenty-four hours there are so many polynuclears that when the toxines begin to escape from the circulation they engage the bacteria in more active warfare and often make quick work of them. This matter of polynuclear leucocytosis is overlooked by many. Operators often get the pus out in a very dangerous manner, pulling out the bowels, besides washing out the peritoneal cavity and doing other work that results disastrously to the patient; many surgeons, with the best of intentions, disable the reparative powers of their patients. These patients cannot stand such extensive work. The surgeon who takes out the bowels, handling them a great deal, washing them, and replacing them in the abdominal cavity, throws many a patient into the condition of shock. Finney, of Baltimore, reports some successful cases; but I do not think this is the operation that should be carried out. I believe that we should shock these patients as little as possible; that we should get the pus from the peritoneal cavity through small incisions, and the toxines from the blood with the aid of saline transfusion. All separate collections of pus should be broken down. Do this expeditiously, and it is not necessary that the bowels should come much into view. The importance of an established hyperleucocytosis is very often overlooked. If I operate upon a case of general suppurative peritonitis and prick my finger, I die, yet the patient lives. Now, why is this? Because, in suppurative peritonitis hyperleucocytosis is established. The polynuclears are there to fight and kill the bacteria, and so save the patient's life. But in the case of a pricked finger the bacteria get too good a start, grow too rapidly for the polynuclears. It is a question of which foe is intrenched.

DOUBLE INGUINAL HERNIA.

Case V.—This young girl has a double inguinal hernia. I shall operate on one side while my assistant operates on the other. It is important, for cosmetic purposes, that the incisions should be symmetrical. With a pair of scissors I cut through the skin and fascia, exposing the external oblique aponeurosis, which I now split with my fingers, and the hernia bulges out. I am looking for the ilio-inguinal nerve, which should be gotten out of the way, or else the patient may complain of pain afterwards. I do not mind sacrificing this nerve, for it supplies only a small portion of the skin. The hernial sac is next brought into view and stripped of its attachments. The peritoneal cavity is then opened, and here is the round ligament presenting; it is displaced, and I shall put it where it belongs. Everything is now exposed. Here are Poupart's ligament, the inguinal canal, etc., and lying in the canal of Nuck is the round ligament and the fold of peritoneum which accompanies it. As you know, this pouch of peritoneum which follows the round ligament along the inguinal canal is analogous to the processus vaginalis in the male. The internal oblique and transversalis muscles are next brought together beneath the lower reflexions of the ligament by means of strands of chromicized catgut (which last four weeks) and tied. As a final procedure I shall reattach the round ligament. This peritoneal sac is an abnormal one and has adhesions, not the result of any inflammatory process, but as a congenital defect. The round ligament is now hooked up and held where it belongs. Next, I shall restore the continuity of the canal in the usual method, by suturing the external oblique aponeurosis over the round ligament.

In closing the skin wound I shall use subcuticular sutures, in order to avoid stab cultures of the *Staphylococcus albus*. The fat is held together by atmospheric pressure; this fact is so important that it should be kept in mind. Atmospheric pressure approximates the fat much better than any method we may use. Before closing the wound I will flush out with saline solution in order to displace any air that may be present in the wound. This subcuticular suture leaves practically no scar. Aristol is next applied, then a pad and gauze. This patient will be kept in bed eighteen days, although De Garmo allows them up in twelve days. I prefer leaving them quiet a little longer.

Marcy, of Boston, seals the wounds with iodoform and collodion, and it is a very good way. I have used it, and the wounds healed kindly and almost without any scar. In using the subcuticular suture any scar that may be left will fade, and at the end of two years one would not realize that an operation had been performed.

This condition of double inguinal hernia in a woman is rather rare.

I shall now direct my attention to the prepuce, which adheres to the glans clitoridis. As I strip it back, notice the glans bob out. The next thing is to do a circumcision. These preputial adhesions in girls cause the same symptoms as in boys, Nocturnal enuresis, reflex disturbances, such as epileptoid attacks, etc., may be relieved by circumcision after breaking up any adhesions that may exist. Preputial adhesions are more common in girls than in boys. They are frequently a cause of local eczema and other results of irritation. Pruritus about the vulva and excoriations may exist, due to decomposition of the smegma beneath the preputial adhesions. It is chiefly in young girls that the best results are obtained from stripping away these adhesions. Since the adhesions may reform, it is the best plan to take up the prepuce with a pair of forceps and snip it off, apply an aristol dressing, and then let it alone.

DOUBLE OSTEOTOMY OF HIP FOR DEFORMITY.

BY J. MACKENZIE BOOTH, A.M., M.D.,

Lecturer on Clinical Surgery in the Children's Royal Infirmary, Aberdeen, Scotland

---

In the following narrative I have endeavored to describe an attempt by surgical and orthopædic means to relieve an exceedingly severe and unsightly deformity that resulted from strumous disease of the lower extremities.

The subject of this deformity, J. F. E., aged thirty-one years, was principal in a city grocery business when first seen, but had been born and bred in a remote and mountainous district of Aberdeenshire, Scotland, and only for the last few years had been resident in a city.

He first came under my notice in February, 1896, when suffering from a boil on the hip, after which he asked my advice regarding his deformity. I found angular deformity, with rotation of both hip-joints, obliquity of pelvis, or scoliosed spine, and shortening of the right leg present, and proposed surgical interference, but suggested a confirmatory consultation with another surgeon in the first place. Accordingly, Professor Ogston saw him with me, and after examination recommended osteotomy of both hips. At that time, however, his business arrangements did not permit of his undergoing an operation, and it was not till two years later that he was able to arrange for its performance.

On the 7th of March, 1898, he presented himself for admission at the Aberdeen Royal Infirmary, when the following history was elicited. He stated that he was the second of a family of eight, none of whom was strong, while more than one had suffered from tubercular disease. His father and mother, however, were still alive and healthy, aged, respectively, seventy-five and sixty-five years.

As a child the patient had suffered from a lung affection following measles, which kept him weakly for a long time. When nine years of age, during a very hot summer, after overheating himself, he suffered from a painless intestinal affection accompanied by jaundice and great weakness, for which he had to lay up, and this was the commencement of a rest in bed for three years. After a month he began to experience a tired feeling in his right leg, which soon became a dull, gnawing pain about the top of the thigh bone. He found it gave him most relief if he lay on his left side with his right leg flexed slightly and adducted with rotation inward, and so resting on his left leg with the pelvis heightened. He had pain if he attempted to lie with his back straight along the bed, as he was wont to do. Marked wasting of the tissues in the gluteal region ensued, and soon a zone of swelling and heat could be marked off round the hip-joint. The zone pointed, an abscess burst over the trochanter major, and from it thick yellow matter was discharged. Simultaneously with this he had bed-sores in various parts, worst in the sacral region and in the heels of both feet, and his femoral and inguinal glands were swollen. When the discharge had gone on some time from the right thigh, the pain lessened, but the loss of motion in the limb still continued; and in addition pain set in all down the left side of his body, as if caused by lying thereon. His shoulder was affected, and after being hot, painful, and swollen for some time, suppurated like the right hip. His left elbow stiffened very much, but did not discharge. Suppuration also took place from bed-sores over the lumbar region of the spine. Over the left hip, which had assumed the position of flexion and abduction, a swelling appeared, which was opened and continued to discharge for some time. The right leg at the same time became affected below the knee. Suppuration took place, and eventually, under chloroform, a piece of bone was extracted.

In addition to these named regions there were little localized abscesses all over the body. As time went on his condition improved. His left elbow and left knee, which had been stiff, regained somewhat their normal movements, but his left hip did not improve, and became quite stiffened. The right hip had movement only to a limited extent. The patient also found that his pelvis and lumbar spine had lost their natural position, and that there was marked shortening of the right leg. During all this time he had

little pain, but night-startings from the bones rubbing on one another, especially at the hips. After about three years he rose with his left hip-joint completely useless and his right one little better.

Physical Examination.—The patient is not strong in appearance, although his body looks plump and well nourished. Over various parts of the body and even in the face the cicatrices of former sores are seen. His right leg seems to be shorter than his left, and the pelvis is twisted to the side and somewhat backward on the right side. The accompanying illustration shows him leaning against a wall with the help of a stick in his left hand.

I. *Lower Limbs*—(*a*) *Right Side.*—The foot is hyperextended and cannot be flexed normally. Lateral movement is slight. The knee is twisted to the inside, so that the patella looks forward and inward. The patella seems flattened with more defined angles than usual, and inside the inner condyle of the femur is a deep depression. The leg shows a long red scar in front, whose long axis is bent with the concavity inward. The patient can flex the leg to a little over a right angle. The hip shows a scar over the trochanter region. There is no marked wasting. The extensor aspect is not so well developed as in the left limb. There is a deep pocket in the gluteal region. Movement here is limited to flexion and extension only through an arc of forty-five degrees. Little or no abduction and adduction. Rotation impaired, more especially rotation inward.

(*b*) *Left side.*—The foot is almost normal, but slightly inverted. The knee is normal, but flexion cannot take place to the usual extent. The inner tuberosity of the tibia is prominent.

Hip.—No wasting to be seen, but there is complete ankylosis to the pelvis, which moves on trying to extend or flex the hip. A deep pocket exists in the gluteal region. On the whole, the left leg looks distinctly sounder than the right and shorter one.

Measurements.	Right side.	Left side.
Anterior-superior spine to internal condyle of femur . . .	13 inches.	13¼ inches.
Anterior-superior spine to internal malleolus	26 "	27¾ "
Internal condyle to malleolus	13 "	14½ "
Circumference seven inches above internal condyle	17¾ "	18 "

II. *Upper Limbs.*—The right upper limb is well developed and shows no loss of movement. The left one is much scarred in the

FIG. 1—Appearance of patient when dressed.

FIG. 2.—Anterior view

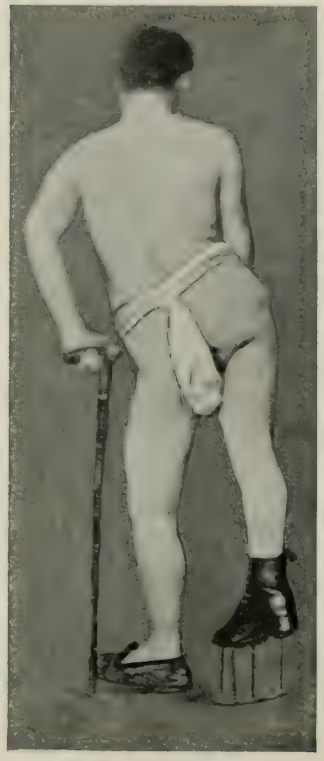


FIG. 3.—Posterior view.

Dr. Booth's case of double osteotomy of hip for deformity.—Before operation.

FIG. 5.—Appearance of patient when dressed.

FIG. 6.—Anterior view.

FIG. 7.—Posterior view.

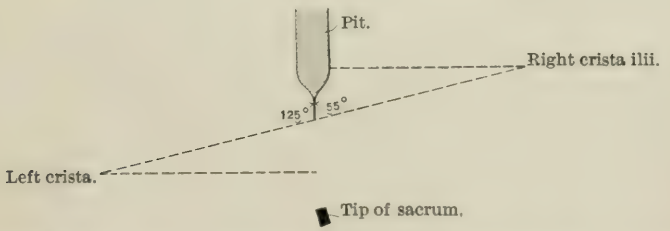
Dr. Booth's case of double osteotomy of hip for deformity.—After operation.

region of the shoulder, and the movements are impaired. He cannot strike a straight blow before him, nor raise the arm above his head, and abduction is not present in normal measure. The elbow is not perfect. The olecranon process is very prominent and movement is impaired. He cannot fully extend the joint, and pronation and supination can only be effected with much movement and accommodation of the humerus and shoulder-joint.

III. *The Trunk.*—The right shoulder is markedly higher than the left. The spine in the dorsal region is scoliosed slightly to the right side. On trying voluntarily to level the shoulders, the spine in the lower dorsal and lumbar regions inclines and bends much to the left. There is a deep pit with lordosis in the lumbar region. The pit is about three inches or four inches in length, and in its depth the hard vertebral processes are felt. It appears to extend from the lower dorsal to the end of the lumbar vertebræ. The lower end of the spine with the sacrum inclines to the right rather abruptly, and the lower end of the sacrum stands out as a prominent knob. Fig. 3 shows the condition of the trunk viewed from behind while patient attempts to stand against a wall.

The pelvis is twisted to the right and slightly backward on that side, with the patient lying on his face. A line drawn transversely from the highest point of the crista ilii on the right side crosses the pit about the junction of the middle and lower thirds. Such a line drawn from the left side crosses the centre of the back about one inch above the tip of the sacrum, which is prominent. A line join-

FIG. 4.



ing the two cristæ crosses the spine one inch below the pit, forming with the vertical plane an angle of one hundred and twenty-five degrees on the left and fifty-five degrees on the right side. (Fig. 4.)

Locomotion can only be effected slowly and with much difficulty By means of two walking-sticks and a long iron patten attached to

the right boot a very tortuous, corkscrew-like, and tardy progression is attained, which naturally attracts the attention of the passers-by. Nor can he go far at a time without getting tired out. Otherwise his general health is good, the respiratory, circulatory, genito-urinary, digestive, and nervous systems showing no abnormality.

On the 16th of March, with due antiseptic precautions, subcutaneous division of both hips was performed. Both legs were pulled and rotated into as good a position with reference to the trunk as possible, and a strong plaster casing applied to both thighs, to the pelvis, and to the lower part of the abdomen. He was then kept in bed for two months, during which time there was difficulty from chafing of the skin and weakening of the plaster, requiring the use of cotton-wool packing, zinc ointment, extension by weights, and the employment of a Lister splint.

On the 13th of May the patient was allowed to get up for a short time, but was unable to put his whole weight on his legs till the 26th of June. On the 6th of July he was able to be out for a little while with the aid of crutches.

The measurements now were as follows:

	Right.	Left.
Anterior-superior spine to internal condyle	14 inches.	16½ inches.
Anterior-superior spine to internal malleolus	27 "	29½ "

He has also gained three inches in height, measured standing on both feet, the shortened right leg being fitted with an extension boot. During July he had a slight recurrence of the tibial inflammation, and a small abscess formed, discharging about a drachm of thick yellow pus.

He left the hospital on the 29th of August, when he was able to walk a short distance with the aid of two walking-sticks, or a stick and a crutch. On the 9th of September he left for the country home where he was brought up, and there he remained till November, 1899, the date of this communication. During that time, with the exception of three weeks in January, 1899, and three days in October, he gradually grew stronger, walking at first with two sticks and afterwards with one, until he could cover over three miles at a stretch.

Figs. 5, 6, and 7, from photographs taken at the time, show the change in his appearance eighteen months after the operation.

In remarking on the foregoing case the general conditions were distinctly unfavorable. The family history, the previous ill-health, and the marked tendency to bone disease all contraindicated successful operative procedure. Yet the deformity was so unsightly, and locomotion was so ungainly and difficult, that the surgeons unhesitatingly recommended, and the patient readily welcomed, the hope of relief by operative treatment. As was to be expected, the convalescence was protracted and broken by repeated recurrence of the tibial inflammation. Skiagraphs were made of the pelvis before and after operation, which showed indifferently the ankylosis of the left and the distortion of both hips and their improved position after treatment. The results have been in the main distinctly satisfactory. The appearance, gait, and power of locomotion of the patient have all been favorably modified, and with the exception of a halt, due chiefly to the ankylosis of the left hip, there is very little noticeable peculiarity. The use of the O'Connor extension apparatus, too, instead of the unsightly iron patten, has aided considerably in the improved appearance effected by the operation and conduced to greater comfort in locomotion.

SOME OF THE MORE REMOTE EFFECTS OF FRACTURES, WITH ESPECIAL REGARD TO THOSE PRODUCING DEFORMITY AND LOSS OF FUNCTION.

CLINICAL LECTURE DELIVERED IN THE CHARITY HOSPITAL, NEW ORLEANS.

BY EDMOND SOUCHON, M.D.,

Professor of Anatomy and Clinical Surgery in the Tulane University, New Orleans, Louisiana; Fellow of the American Surgical Association.

GENTLEMEN,—The remote results of injury form a most interesting, practical, and important chapter in the history of fractures, and have occasioned more suits for malpractice than all other surgical conditions put together, on account of the deformity and loss of function which may ensue even in spite of the most skilful treatment. In a general way, the following remarks apply especially to conditions met with in adults. In children, fractures with deformity and impairment of function sometimes correct themselves to a wonderful extent: in many cases it is almost impossible to determine the point at which the fracture took place. In old people, on the other hand, there is little or no tendency to amelioration.

Let us take up systematically the different varieties of fracture, noting the more remote results that may follow.

Fractures of the Skull.—Fractures of the vertex, even when there is no displacement of the fragments, may be followed by grave cerebral disturbances due to the accompanying concussion of the brain. Any cerebral injury sufficient to produce unconsciousness should be treated by absolute rest in bed and subjected to careful supervision, particularly to detect the symptoms of slowly oncoming hemorrhage from the anterior or posterior branches of the middle meningeal or the middle cerebral arteries, the symptomatic significance of a period of consciousness between two intervals of unconsciousness being always noted as of special importance. Unfortunately, we are too often dependent upon the observation of

the laity for such a history. The diploe being spongy, the external table may be driven into it without affecting the inner table, which in turn may be depressed into the cortex without visible external injury beyond a slight contusion. Localizing symptoms are of the most vital importance in these cases, for a severe blow upon the vertex may result in fracture, not *in loco,* but at the base of the skull.

When fragments of bone are driven into the cerebral substance, inflammation, sepsis, vagaries of mentality or of disposition, violent cephalalgia, or even epileptiform convulsions may ensue; and, if they are not promptly removed, a brain-habit may be established, against which later operative measures may be powerless. All impingements of bone causing pressure upon the brain substance should, therefore, be promptly removed in every case in which the site of lesion can be determined and is accessible. The trephine-opening should be sufficiently large for thorough exploration, as it will in time diminish; but, filling up mainly with fibrous tissue, the brain is liable to injury at this point; additional security may be obtained by the use of a silver plate.

Fractures at the base of the skull are often, though not always, fatal. If death does not follow immediately, the greatest danger is from septic infection from the nasal, buccal, pharyngeal, or aural canals, which should constantly be sprayed with boric solutions. Bone, blood-clots, callus, or inflammatory exudates may press upon one or more of the twelve cranial nerves, and produce various local anomalies whose anatomical basis can in many cases be determined by careful study of the symptoms. Doubtless many fractures at the base escape notice entirely.

Fractures of the nasal bones are often accompanied by distressing deformity, notwithstanding the most skilful treatment. So rapidly do repair and union take place that unless reduction be made within forty-eight hours it may be exceedingly difficult to accomplish. Sometimes a fairly good result may be obtained by passing a Mason drill beneath the fragments, parallel with the transverse axis of the face, and exerting pressure from before backward by means of a rubber band. In fracture of the septum careful search should be made for deviations, which may be corrected by the Roberts method with a harelip pin.

Fractures of the malar bone and of the body of the superior

maxilla are always due to severe blows, as from machinery or a heavy fall. Owing to the alteration in the personal appearance and the impingement upon the cavities of the nose, orbit, antrum, or even of the mouth, reposition is imperative, and may be effected either through an external wound by means of a strong hook or a *tire fond,* or from within the oral cavity by pressure upon the zygomatic arch, by entering the maxillary sinus just above the first molar tooth, about three-quarters of an inch below the inferior margin of the orbit, and making pressure and traction from within outward.

Fracture of the alveolar process of the superior maxillary may interfere with mastication by disturbing the adjustment of the "bite," even though the union may be perfectly firm; this is often forgotten. Perfect reposition, held by proper interdental splints or appropriate special apparatus, will give satisfactory results, the patient meanwhile being fed with liquids by a tube introduced back of the last molar teeth.

Fractures of the Inferior Maxilla.—Fractures of the lower jaw usually occur near the dental foramen, rarely at the epiphysis. The neck of the condyle may also be broken by a blow upon the chin. If bilateral and near the symphysis, considerable difficulty may be experienced in securing the fragments. Accurate coaptation is of supreme importance, as imperfect adaptation of the teeth to those of the upper jaw may result in difficult or painful mastication, or indeed may prevent the adequate mastication of solid food altogether. We cannot urge too strongly the examination of injuries within the oral cavity, as not infrequently fractures have been poulticed or otherwise treated as of inflammatory origin. If the fracture is compound by a wound of the mucous membrane, infection may result and caries and necrosis follow. The pus cavity must be drained, any sequestra removed, and later the fragments wired if indicated. As far as possible, operations should be intra-oral, cicatrices upon the skin being likely to become adherent to the bone, depressed, and, by virtue of their position, unsightly.

Fractures of the Vertebræ.—The importance of these fractures depends upon the concomitant injury of the spinal cord or the emergent nerves, the symptomatology being largely an anatomic study. Fractures of the atlas or axis are usually, though not always,

immediately fatal, below this the prognosis being relatively more favorable as we descend. Death occurs from exhaustion. Below the tenth dorsal vertebra, injuries may result in imperfect forms of paralysis of motion and sensation, due to the cauda equina allowing separate strands to be impinged upon. Simple fracture without displacement is somewhat difficult to detect. If accompanied by displacement or by dislocation of the entire vertebra, it is usually more easy of recognition.

Fractures of the coccyx with displacement may be followed by coccygodynia (painful coccyx). Rebreaking and resetting or complete removal of the offending portion is indicated.

Fractures of the larynx are produced by criminal efforts at garroting, the deformity being an anterior projection with depression inward of the lateral portions of the cartilages. Dyspnœa and bloody expectoration are absolute indications for preliminary tracheotomy. The fragments being carefully replaced by manipulation, the patient is placed in bed in the recumbent posture, with sand-bags on both sides of the head.

Fractures of the clavicle are, next to those of the radius, the most common of fractures. When within the ligaments at either extremity of the clavicle, they are attended by no deformity; but beyond the ligaments in either direction deformity is the rule, though in children an intra-periosteal fracture may be so slight as to escape notice, and by the time the patient has become an adult it may have entirely disappeared. In old persons a false joint is not uncommon. Deformities of the clavicle following fracture do not impair the function of the limb unless they exert pressure upon important nerve-trunks.

Fractures of the Scapula.—Fractures of the acromion process may result in permanent deformity, and that of the glenoid process is often followed by functional disability. Fractures of the coracoid process, or of the "surgical neck of the scapula," are rather rare. Fractures of the body can be produced only by such an accident as being run over by a carriage. In all of these the fragments may be kept in place by pads with adhesive strips, together with a Velpeau bandage.

Fractures of the Humerus.—Fractures of the anatomical neck, especially of the stalactite form, or fractures of the tuberosities, may result in restricted usefulness of the arm. In fractures of the

surgical neck, displacement occurs almost invariably. It is by far the most important of the fractures of the upper extremity of the humerus, though perhaps not quite so frequently met with as those of the lower third of the bone. The tendency of the lower fragments is to be drawn upward and inward by the pectoral and flexor muscles, the upper end of the upper fragment being drawn upward, outward, and backward by the supra-spinatus and infra-spinatus and teres minor muscles. If union takes place in this position, an angular deformity results. Fractures just below the deltoid are particularly prone to this complication, and require a special mode of treatment, with the arm abducted for ten days till the spasm of the deltoid muscle has been overcome. More important than the deformity, which is seldom accompanied by loss of function, is injury to the adjacent structures. The axillary or brachial artery or vein may be so bruised as to form a thrombus, followed by gangrene of the extremity; the arterial coats may be so injured as to give way and produce an aneurism, or the artery itself may be completely torn across. The musculo-spiral nerve, in consequence of its lengthened and close relation to the bone, is particularly liable to injury. Fractures of the lower extremity of the humerus are likely to result in deformity in inexperienced hands by being set in an extended position, resulting in ankylosis in a bad position which may require refracture and resetting, or the formation of a pseudo-articulation. Reduction is made by traction upon the flexed forearm, with a complete replacement under ether if necessary; then lint with lead-water and laudanum should be wrapped about the joint and an anterior rectangular splint with a posterior moulded trough applied, the hand being then in marked supination. A weight attached to the elbow will prevent shortening. Lateral angular displacement outward (cubitus varus) is one of the deformities to be avoided. In these injuries, even if no ankylosis occurs, the range of motion may be restricted by callus formation.

Fractures of the Ulna.—Fractures of the olecranon process may result in fibrous union, though bony union may occur. Non-union is sometimes met with. The mechanical loss of strength in extension is without remedy, though wiring under antiseptic precautions has been attempted. The best results are obtained by holding the fragments down with a pad and figure-of-eight loops

of adhesive plaster, with an anterior splint having a very wide obtuse angle (almost straight). Passive motion must be made with extreme caution, and not too early. Fracture of the coracoid process usually occurs about a quarter of an inch below the apex. There is some danger of restriction of motion of the forearm, due to excessive formation of callus. The forearm should be held in extreme flexion upon an anterior angular splint. Fractures of the shaft and lower extremity of the ulna are easy of diagnosis because of the prominence of the longitudinal edge upon its posterior surface.

Fractures of the Radius.—Fractures above the bicipital tuberosity are exceedingly rare, the absence of rotation of the head being detectable by forcing the thumb posteriorly just below the external condyle, at the same time rotating the forearm in the long axis of the bone. The treatment is the same as that of a break in the coracoid process of the ulna. Fractures of the shaft of the radius below the bicipital tuberosity and above the insertion of the pronator radii teres ("upper third") are liable to result in marked deformity, the lower fragment being drawn towards the ulna by the pronators, the upper fragment being rotated backward and inward by the biceps, necessitating forcible retention of the forearm in the flexed and supinated position. Fractures of the shaft may result in impairment of the interosseous space requiring separation of the union by chisel and hammer. Fractures of the lower extremity of the radius are three in number: (1) "Barton's," a chipping off of the posterior articulating surface; (2) Colles's, occupying from a quarter of an inch above the articulation to an inch and a half above; (3) Smith's, one and a half to two and a half inches above the joint; above that the lesion is called a fracture of the shaft. Colles's fracture is the most frequent fracture in the body. It may be produced in one of three ways: (1) by a fall upon the heel of the hand, the force being transmitted through the carpus to the radius,—the ulna escaping because it does not enter into the articulation; (2) a fall upon the anterior distal; (3) or upon the posterior distal extremity of the metacarpus,—the break then occurring by a cross-break strain at the point of attachment of the radio-carpal ligaments. Firm union may be expected within a month, an ununited fracture being here almost unknown, which may account for the success following the

vagaries of treatment of various surgeons. Even those fractures which cause no immediate deformity may be followed by a general thickening of the tissues about the wrist, due to infiltration of the sheaths of the tendons without inflammatory exudates, which may persist for years. Passive motion should be begun at the end of the second week, and after union has taken place should be persisted in. In many cases marked deformity is inevitable, but may be attended by little or no disturbance, a much deformed wrist often being apparently as useful and as strong as its fellow.

Fractures of the Carpus, Metacarpus, and Phalanges.—Fractures of the carpus are usually due to direct traumatism, and may be accompanied by general swelling with stiffness of the wrist unless very carefully treated. Fractures of the metacarpus and phalanges may cause permanent deformity without functional trouble, though stiffening of the tendons may follow from adhesion of the tendons to their sheaths.

Fractures of the sternum with inward displacement may press upon the structures of the mediastinum. The fragments should be replaced by means of a strong hook or a screw elevator; resort to the trephine may be necessary.

Fractures of the ribs seldom give deformity, but may cause discomfort from a stitch in the side or from intercostal neuralgia.

Fractures of the crest of the ilium may go undetected even when there is displacement. Fractures of the pelvic bones depend for their significance upon the injury to the contained viscera, particularly the bladder or the urethra.

Fractures of the Femur.—Fractures of the neck of the femur are not infrequently followed by permanent neuralgia and some claudication. It should not be taken for senile atrophy of the head of the femur. In the aged the intra-capsular variety of fracture seldom, if ever, unites by bony tissue, and those who, on account of excessive corpulence or depressed vitality, cannot be readily handled or moved about in bed are prone to suffer from bed-sores in spite of the greatest care and the use of air-beds or water-beds; the digestion is impaired; hypostatic congestion of the lungs may occur, and the patient succumb from exhaustion. There is much less danger from these complications when the patient can be got out of bed or has a greater degree of vitality. The early history of fracture of the neck is so diagnostic that

error would seem impossible; but the slight traumatism applied to the foot and the slight primary deformity may mislead a superficial observer.

Fractures of the shaft of the femur almost always result in some shortening which is unpreventable. If the shortening exceeds three-quarters of an inch, the patient will limp; in this condition the use of a thick cork sole to the shoe is to be recommended. In some carelessly treated cases there is inversion or eversion of the toe, for which refracture and resetting are the only remedy. Fracture of the lower extremity is almost always followed by impairment of function and deformity, especially if the case has not been seen early or has been injudiciously treated. The prognosis is always guarded, as involvement of the joint may coexist with sepsis, suppuration, or ankylosis.

Fractures of the patella seldom unite by bony union, even in healthy adults. If there is much separation of the fragments, which depends upon the degree of laceration of the involucrum, there will be a dragging of the great toe, due to the loss of power in extension by the quadriceps extensor muscle. There is also difficulty in descending steps, by which stout persons are more inconvenienced than thinner people. Many cases of wide separation of the fragments retain their function by reason of the integrity of the sheath or involucrum, the patella in reality being only a sesamoid developed in the tendon of the quadriceps to give additional leverage.

Fractures of the Tibia.—Fractures of the tuberosity of the tibia may be followed by permanent deformity and impaired usefulness of the limb. Fracture of the shaft may not be followed by overriding of the fragments, but, even when this occurs and causes a constant limp, radical operative measures are seldom justifiable. Fracture of the internal malleolus will determine a synovitis of the ankle-joint with some subsequent stiffness, which is more serious when the fracture is compound. When associated with Pott's fracture there often remains a more or less permanent deviation of the foot with so much pain in walking as to require active surgical interference.

Fractures of the Fibula.—Fractures of the upper extremity of the shaft of the fibula are but seldom followed by deformity or functional disturbance, though somewhat difficult of diagnosis on account of the absence of displacement and the thickness of the

peroneal muscles. Fractures of the lower extremity (Pott's fracture) are of extreme importance. Too much care cannot be exercised in their diagnosis and treatment. Every injury about the ankle should be examined for fracture, and if there is any doubt should be treated as such. The fracture is often mistaken for a sprain, and the angular deformity it occasions is progressive, even after union has taken place. Fractures take place at three points, —(1) through the lower three inches of the fibula above the external malleolus; (2) through the outer articular surface of the tibia; (3) through the lower extremity of the tibia above the internal malleolus. If reduction is thoroughly made and thoroughly maintained the prognosis is favorable, though pain on motion will even then persist for a considerable time. If there is either outward or backward displacement, the weight of the body is thrown away from the support of the ankle-joint and pain on walking results, with fatigue upon walking any distance.

Fractures of the tarsus are usually the result of crushing violence, excepting that of the tuberosity of the calcaneum which may follow a too violent contraction of the tendo Achillis. Ankylosis of the joint involved may follow any of these fractures, a cautious prognosis being always given. Fracture of the tuberosity of the os calcis is treated with the familiar slipper and ring; there may be fibrous union resulting in a loss of power and a dragging of the foot. All other fractures of the foot are treated with a plantar splint and fracture-box, in the flexed position.

Résumé.—The fractures which are oftenest accompanied by deformity are those of the vertex, nose os malæ, superior maxilla, coccyx, clavicle, surgical neck and shaft of the humerus, condyles, lower extremity of the radius, carpus, sternum, neck of the femur, shaft and condyles of the femur, tuberosities and shaft of the tibia, Pott's fracture, and fracture of the tarsus.

The fractures which are oftenest followed by impairment of function are those of the vertex, base of the skull, nose, alveolar process of the superior maxilla, all parts of the lower maxilla, vertebræ, coccyx, acromion and glenoid process of the scapula, the head and condyles of the humerus, the olecranon and the shaft of the ulna, the radius, ribs, pelvis, the neck and condyles of the femur patella, tibia, and fibula, Pott's fracture, and fracture of the tarsus.

Obstetrics and Gynæcology

CYSTOMA OF BOTH OVARIES; HEMORRHOIDS AND EXTRAORDINARY DILATATION OF THE RECTAL VEINS; LIPOMA OF THE URETHRA; LARGE LABIAL TUMOR; LIPOMA OF THE LABIUM MAJUS.

BY ALEXANDER J. C. SKENE, M.D.,
Of Brooklyn, New York.

CYSTOMA OF BOTH OVARIES.

This case is of especial interest on account of the difficulty of making a positive and complete diagnosis.

The patient, a young maiden lady, enjoyed good health until about a year ago, when she began to suffer from attacks of pelvic pain, quite acute in character, but not sufficiently severe entirely to disable her. She was never quite free from it, and always suffered more at her menstrual periods. There was also disturbed digestion, and about four or five months ago she noticed an increase in the size of the abdomen. When I saw her, about a month since, she was decidedly anæmic, had lost considerable flesh, was rather emaciated, and had continuous fever,—evidently septic. Her temperature remained continuously at about 100° F., rising a little at times in the afternoon and evening, but never coming down to normal. She also developed a cough, which she attributed to a cold, but I was unable to find evidence of any bronchial or pulmonary trouble, or of any cardiac anomaly, except weak circulation and rapid heart action. I made every effort to ascertain the cause of her fever, and believe it to have been due to sepsis of a mild form,—subacute sepsis, if that expression is permissible. During the last month of her

illness and for some time previously she suffered from an exceedingly annoying irritability of the bladder, being unable to retain its contents for more than an hour or so at a time; this added much to her discomfort and her ill-health by disturbing her rest so frequently at night.

On physical exploration I found two cystic tumors of the abdomen, one on each side, extending on the right side two inches and on the left about one inch above the umbilicus. While the two masses or cysts were well apart above, they came close together below and descended deep into the pelvis, crowding the uterus forward and upward. This condition accounted for the irritability of the bladder, which was so hemmed in and pressed upon that it could not be distended. The best idea that I can give of the mass of tumors is, to liken it to an immense uterus bifundalis unicornus, the cervix filling the pelvis almost completely and extending nearly to the pelvic floor. The tumor or tumors were cystic, and they were so thoroughly united below that they appeared to have a common origin, bifurcating above. The uterus was firmly fixed to the tumor, of which it appeared to form a part; it could not be moved independently of, or be outlined from, it, although the case was a favorable one for examination on account of the patient's emaciation and lax abdominal muscles. On internal measurement the uterus was not found enlarged. The lower portion of the tumor was so rigidly attached in the pelvis that it could not be moved in the slightest degree. The upper portions of the tumor were distinctly and clearly cystic, and I concluded that the case was either one of multiple intraligamentous cyst, or else there were two cysts, one in each ovary, united by adhesion in the median line where they came together in the pelvis, and that the two, if two there were, were adherent to the sac of Douglas all around and to the uterus. Of the two conditions, I thought the double ovarian cystomata the more likely to give rise to the physical signs mentioned. On operating I found an ovarian cyst on each side. The cysts had formed firm adhesions early in their development deep down in the sac of Douglas, and they had become firmly adherent to each other and to the entire posterior wall of the uterus. Beginning necrosis was also present in both cysts, so that it was impossible to tap them, because of the friable condition of the cyst walls. They were really hæmatocysts, for they were both filled with blood, thick and tarry in consistency. After

emptying them and definitely settling the question of adhesions in the sac of Douglas and to the uterus, I endeavored to separate the two cysts from each other and from the uterus, but was unsuccessful. I noticed also a marked infiltration or enlargement of the utero-ovarian ligaments, each of which was nearly as thick as my little finger even close to the uterus. I also found it impossible to separate the cysts from their deep pelvic adhesions, on account of the commencing necrosis. They were so friable that the slightest traction on the cyst walls caused them to break down. I determined at once that it would be safer and easier to remove the cystic ovaries together with the tubes and the uterus. I succeeded without trouble by dividing one broad ligament and separating the adhesions in the pelvic cavity from below. In the deeper portion of the bifurcation of the two cysts, posteriorly, there was an extensive intestinal adhesion, which was successfully treated in the usual way.

Fig. 1 shows the several points of union of the cysts and uterus.

Another peculiarity that I am reminded of by looking at the specimen is the freedom of the Fallopian tubes from adhesions to the cysts of the ovaries. I laid the uterus open for the purpose of showing that it was not enlarged and that it was firmly adherent to the cysts.

Fig. 2 shows the point at which I tried to separate the adherent uterus. The bleeding was such that I was obliged to abandon the attempt. The union of the uterus and the cysts was so intimate that there was no line of demarcation for cleavage. Of course, the cysts are shrunken and are very much smaller than they were originally.

The patient made an easy and complete recovery.

The operation was done according to the method of Dr. L. Grant Baldwin, surgeon to St. Peter's Hospital, Brooklyn. Just here it should be stated that I adopted Dr. Baldwin's method of doing hysterectomy when he devised it over three years ago, and have practised it ever since, my only modification of his technique being the use of the hæmostatic forceps instead of ligatures to close the vessels. For the benefit of those who are not familiar with this method, I quote from the doctor's account of the operation:

"For a long time I felt that the way to do hysterectomy of this kind was to have a forceps of sufficient strength and of such shape that you could put it on the broad ligaments down to and including,

if possible, the uterine artery and its branches on each side, and cut the tumor off, and then do the other work afterwards. I have now done four abdominal hysterectomies in that way by simply clamping each broad ligament with the forceps and then cutting on top of them down to the uterus; then making a flap anterior and posterior, then taking another forceps and putting it on the other side and cutting it off immediately, without any ligatures, getting the uterus out in six or seven minutes without any trouble at all. This has enabled me to tie the arteries separately, having absolutely no ligatures exposed when the peritoneum is closed up, except the over and over suture of the catgut used in sewing the broad ligaments. The tumor is all out of the way, the pelvis is clean and clear, and you can sew up the broad ligaments and do your work with the greatest ease."

HEMORRHOIDS WITH EXTRAORDINARY DILATATION OF THE RECTAL VEINS.

The patient, a widow, aged forty-eight years, has had three children and two miscarriages. In 1875, during her first pregnancy, she had slight rectal hemorrhages at stool. Under a physician's orders she took injections of alum and doses of tannin, and thinks that this treatment lessened the loss of blood. Large external hemorrhoids developed after the birth of her child; but they were not attended by bleeding. This condition lasted about one week, and then, as the patient remembers, the condition of the rectum became normal and so remained until about six years later.

About 1881 the patient again had profuse rectal hemorrhages that greatly weakened her, but no pain, no external hemorrhoids, or prolapsus of rectum. Astringent suppositories were prescribed by her physician, but without effect; the patient would feel a desire to evacuate the rectum, and at stool would pass clear blood, which would clot immediately and present a "liver-like appearance." The lower limbs became much swollen, the patient being unable to get her shoes on; she was very weak, awakened feeling exhausted, was unable to mount stairs, had fainting spells, complained of constant drowsiness, and became very anæmic and dropsical. She remained in this condition about one year, and then (in August of 1882) was operated upon. The operation was apparently success-

ful, the patient being free from hemorrhages, except very slight ones due (according to the operator) to a small hemorrhoid that had not been removed.

Fourteen months later the third child was born. Within a few months after her delivery the patient had considerable hemorrhage at stool, with much soreness and pain. The physician who had operated in 1882 said these symptoms were caused by fissures. The patient states that she also had prolapsus of the rectum. The doctor prescribed alum injections, and, these failing, performed a second operation (1884), which the patient believes to have consisted of the application of a caustic. For at least a year afterwards the patient was much debilitated, though there was no hemorrhage. Then followed a period in which there was a slight loss of blood when the bowels acted.

In 1892 the second miscarriage took place, but the patient does not remember that it had any marked effect on her condition.

Following this, the hemorrhages increased in quantity and frequency until 1895, when the patient was again operated on. During the latter part of this period she became gradually more and more anæmic and weak, had fainting spells, and the hemorrhoids continued and the prolapsus increased.

The operation performed in 1895 was cauterization, the actual cautery being employed three or four times at intervals of two or three days. Owing to the patient's weakened condition, no general anæsthetic was given, but cocaine was used locally. The patient continued to improve in health and spirits for about two years; she gained in weight and her color became better. In 1898 and the early part of 1899 the bleeding returned and anæmia and exhaustion followed.

This is the history of the case until June, 1899, when, upon the advice of her physician, the patient placed herself under my care.

As already stated, the bowels were kept at rest and the bleeding was controlled by styptic instillations until the time for operation. A laxative was administered on the day before, and in the morning, when we were ready to operate, an enema was given which acted promptly, and with the evacuation the diseased parts were protruded. There was some bleeding, but when the sphincter contracted above the mass of dilated vessels it was controlled. The

parts thus exposed showed an extraordinary varicosity of the hemorrhoidal veins.

Fig. 3 represents the size and general appearance of the protruding mass fairly well. Here and there between the masses of dilated veins were several hemorrhoids of the ordinary kind. The operation was as follows:

The sphincter was dilated, but not paralyzed by over-distention, and the mass from which there was most bleeding was grasped with the long hæmostatic forceps, compressed, desiccated, and then amputated. The other masses—four in all—were treated in the same way. Then the hemorrhoids were removed. The stumps of the largest masses were an inch and three-quarters long, and lay parallel to the axis of the rectum. There was no hemorrhage during or after the operation. Sodium bicarbonate was dusted over the parts, and that completed the operation.

Fig. 4 shows the stumps on one side.

The after-treatment consisted in rest and nourished fluid food. There was no more pain after than before the operation, and so no opium or other anodyne was employed. The bowels were kept quiet for five days, and then moved by a laxative and an enema. There was no bleeding except a very slight oozing, which amounted to a few drops only. The patient gained in strength, continued free from her rectal trouble, and is now in good health.

LIPOMA OF THE URETHRA.

The tumor in this case was situated in the anterior wall of the urethra and projected from the meatus about a quarter of an inch. The patient was young, single, and suffered only from a feeling of fulness and pressure in the urethra, which had gradually become more pronounced and caused discomfort rather than pain. Urination was difficult, but not painful.

A peculiar feature of the case was the entire absence of any subjective symptom excepting the patient's consciousness of the presence of the tumor. Even micturition had not been seriously interfered with. There had been no pain of any moment. Doubtless the only reason in the patient's mind for an operation was the fear that the growth might be malignant.

The tumor was not easily accessible except that part which presented at the meatus. This resembled somewhat an excessive uni-

lateral ectropion, but the use of the probe showed that it was attached to the anterior wall. The mucous membrane covering and hiding the tumor itself was thickened, rough, and quite vascular. Palpation revealed a nodule, sessile yet slightly mobile, occupying the lower third of the urethra. A tentative diagnosis was made of sarcoma, because the history showed that the tumor had grown rapidly during the two or three months immediately succeeding its appearance.

It was determined to remove the tumor by the wire écraseur, so that there might be the least chance of hemorrhage. Upon seizing the presenting part with the tissue forceps, the mucous membrane was torn, and the tumor, being thus in part exposed, was seen to be a lipoma. It was then removed by enucleation. The mucous membrane which formed the capsule was seized with a fine hæmostatic forceps and desiccated; this controlled the bleeding and made a small, dry stump which healed in a few days. The microscopic appearance sustained the diagnosis, and a chemical examination later confirmed it. The tumor was a flattened ovoid measuring an inch in its greatest diameter and being about half an inch thick. It was smooth and solid and had all the appearances of the ordinary fatty tumors found elsewhere.

Fig. 5 represents the protruding tumor as it appeared when first seen.

LARGE LABIAL TUMOR.

The history obtained in this case was that thirty-three years ago the patient had had an abscess of the left labium. It appeared immediately after an otherwise normal puerperium. The abscess was opened and a small quantity of pus was evacuated. The wound healed slowly and left an indurated area about half an inch in diameter which gave no trouble until an active inflammation of unknown origin arose some fifteen years later. The pain was subdued, and the inflammation was arrested after a few weeks by the use of local sedatives. From this time on there was a noticeable increase in the size of the tumor until six months ago, when it had attained the size of two large fists. Since then, and particularly of late, its bulk and awkward situation have caused the patient much discomfort and severe pain.

Strangely enough, while the tumor itself felt sore and sensitive,

the worst pain was not at first referred to it, but seemed to originate in the left ovarian region and to shoot down the left thigh. Locomotion, of course, was difficult; and neither the sitting nor the recumbent position afforded any great relief. Though the patient's general health continued to be fairly good, the worry, pain, and discomfort began so to tell upon her condition that she at last consented to have the tumor removed.

The lines of incision formed a longitudinal ellipse over the most prominent portion of the tumor and approached within an inch of the mucous introitus. By cutting and dry dissection much the larger area was readily uncovered upon the outer side; but the inner mucous investment was dissected from the tumor only with much painstaking care. The vessels as they were encountered were seized temporarily by ordinary artery forceps. When only a long narrow strip of the tumor which contained the large vessels was still adherent posteriorly to the deeper tissues, its separation was completed by means of the electro-hæmostatic forceps. All the vessels were now treated seriatim by electro-hæmostasis. The careful dissection had made it possible to avoid opening the large branches of the bulbus vestibuli. The incision had been so made that coaptation of the edges of the wound by means of salicylated waxed silk was readily accomplished. One suture was omitted from the lower angle to insure drainage.

The after-treatment consisted in protecting the wound from the urine, cleansing the site of the operation with hydrogen peroxide and carbolic acid, and dusting it with equal parts of carbo ligni and dermatol. To prevent the urine touching the wound, catheterization was resorted to for the first few days. Then a urinal tube was employed instead of a catheter. This tube was made of heavy glass sufficiently curved to allow it to be guided into a receiving vessel. A tube six inches in length, one-half inch in lumen, with a two-inch bend and a one-inch lip to fit over the meatus, has been found a convenient size. With this device there was no trouble. Another, but rather inconvenient, method was to let the patient urinate while in the prone position.

The age of the patient, the vulnerability of the tissues, and the contiguity of the mucous surface of the vagina made rapid repair impossible. Nevertheless, the recovery progressed so steadily and

there was such an entire absence of untoward features that the result has been very satisfactory.

Fig. 6 shows the general appearance of the tumor after its removal. The lobulated appearance is suggestive of multiple cystoma, but the tumor proved to be a monocyst.

LIPOMA OF THE LABIUM MAJUS.

When the girl from whom this specimen was obtained was born the nurse noticed that one labium was larger than the other, and called the mother's attention to it. At three months of age the difference in the size of the labia had noticeably increased. From that time until she was eight years old there was no change in her condition. Then the enlargement increased gradually, but more rapidly during the last six months.

The patient was ten years old when first seen, and the appearance of the parts was as represented in Fig. 7. The enlargement was under the skin and superficial fascia, and extended from the lower portion of the labium to the external inguinal ring. On palpation the mass was elastic, lobulated, and quite freely movable under the skin and superficial fascia. In short, the physical signs were the same as those that would be caused by an intestinal hernia which had descended into the labium, except that the tumor was outside of the labium, though so closely connected with it that the two appeared to be one in general contour; and that while the upper end of the tumor extended to the inguinal ring, it could not be traced into the canal. It was, however, difficult to make sure that such was not the case, as the patient was rather stout. There was a possibility of a connection between the tumor and abdominal viscera, and that the portion in the inguinal canal was constricted as in an irreducible hernia. Furthermore, there was no intestinal resonance on percussion; but that only helped to exclude hernia of the intestine, and not hernia of the omentum. There were no positive physical signs upon which a diagnosis could be based, and the history was in no way indicative of hernia of the omentum. By exclusion, the problem was reduced to the question of omental hernia, hydrocele of the round ligament, or lipoma. Hernia was excluded and hydrocele became very doubtful in view of the signs and history and the great rarity of that affection.

Lipoma was most strongly suspected, but a doubt remained, and that doubt became strengthened by hearing that the patient had been examined by a number of well-known physicians and surgeons, and that all of them had declined to make a diagnosis of any kind or to make any suggestions regarding treatment except to advise waiting.

When the skin and fascia were laid open, the diagnosis was plain. There was nothing of interest in the operation except that the tumor was removed without wounding any of the large vessels about the vulva, and that the upper end of the mass extended to the ring but not into the abdominal wall.

Fig. 8 shows the tumor. There is a capsule of fascia surrounding each lobule.

THE ANTERIOR VAGINAL INCISION AS THE ROUTE OF ATTACK IN PELVIC DISEASE; RESECTION OF A CYSTIC OVARY, AMPUTATION OF THE INFUNDIBULUM OF THE FALLOPIAN TUBE, AND SHORTENING OF THE ROUND LIGAMENTS FOR RETROVERSION; MYOMECTOMY THROUGH THE ANTERIOR VAGINAL FORNIX, ETC.

CLINICAL LECTURE DELIVERED AT THE NEW YORK POLYCLINIC, MARCH 24, 1900.

BY J. RIDDLE GOFFE, M.D.,

Professor of Gynæcology in the New York Polyclinic; Visiting Gynæcologist to the New York City Hospital.

Gentlemen,—The first case for demonstration this morning is one in which we find a large retroverted uterus, more or less firmly fixed by adhesions, and complicated by a large, prolapsed, and cystic ovary on the left side. The bulging but elastic tissue in direct contact with the ovary suggests also some involvement of the Fallopian tube. Before beginning the operation upon this patient, I desire to make a few remarks upon the condition here present, and the various methods that have been proposed for its relief.

The etiology is plainly suggested in the history of the case. About a year ago this woman had a miscarriage at the third month; the uterus did not empty itself completely, and for several weeks after the accident, during which time she had no medical attendance, she suffered from a profuse and malodorous leucorrhœa. She has not regained her full strength and has suffered almost constantly since that date from backache, dragging sensations through the pelvis, and pain in the left ovarian region. The menstrual flow, too, has been excessive at each period. We infer, therefore, that a portion of the placenta was not expelled at the time of the miscarriage; that this has undergone suppuration, during which infection, probably sapræmic in its nature, has occurred; the involution of the uterus and its ligaments did not take place normally; the uterus

sagged in the pelvis and finally became retroverted, carrying its appendages with it. From the septic endometritis as a focus, the septic infection has gained access to the left Fallopian tube; has excited an inflammatory process, extending to the fimbriæ and involving the ovary and the peritoneum in its immediate neighborhood. From this circumscribed peritonitis have resulted the adhesions which hold the uterus and the appendages of the left side fixed in this abnormal position.

Inflammatory processes within the pelvic cavity are analogous in every particular to those that occur within the pleural cavity. Both of these cavities are lined with serous membrane, and the only important difference between them from a pathologic stand-point is that the pelvic cavity is in direct communication through the Fallopian tube with the external world, and that on account of the close proximity of the large intestine any morbid process in the pelvis may become infected with colon bacilli. It is, therefore, much more liable to disease, but in both the pleural and the pelvic cavities the inflammation may be serous or plastic. The processes are similar. In the pelvis, however, the plastic exudate, owing to its liability to mixed infection, is much more prone to become permanently organized, whence the frequency of strong and persistent adhesions.

Treatment.—Probably the most popular operation at the present time for the relief of retroversion is the Alexander operation. In appropriate cases this method is successful, but there are some serious objections to it. In the first place, it has a very limited field of application, for the reason that it is indicated in retroversion or retroflexion only when there are no adhesions. Secondly, the round ligaments cannot be found in all cases, and even when discovered they are frequently too fragile to stand the tension that is necessary to carry the uterus into position. Thirdly, the operation necessitates two incisions in the abdominal wall, and is followed by hernia, single or double, in about five per cent. of all cases. It is but fair to state that the field of application of the Alexander operation has been widened so as to include cases of slight and limited adhesion by combining with it an incision in the posterior vaginal fornix, through which the uterus and appendages are set free previous to the shortening of the ligaments.

Ventral fixation is another procedure that has been applied to such conditions as are found in the case before us. This operation

involves an abdominal section, through which adhesions may be broken up, and whatever procedure is indicated by the condition of the appendages may be performed, the uterus being finally stitched to the abdominal peritoneum or parietes at the site of the incision. Dr. Pryor, as you know, freely opens Douglas's pouch in cases presenting the condition here present; breaks up adhesions; restores the uterus to its normal position, and retains it there by an artificial band of tissue, which is developed in Douglas's pouch.

To my mind a much more rational, complete, and satisfactory operation, and one which lends itself to almost every pathological condition in the female pelvis, is the anterior vaginal incision. This is the one I shall employ in the case before us. With the patient upon the back and the knees well flexed upon the abdomen, I grasp the anterior lip of the cervix with traction forceps and firmly but steadily drag it down within reasonable distance of the vulva. As a speculum, you see I use an instrument that I have had modelled after the Simon vaginal retractors, with a hook at the end of the handle upon which I hang a little tin bucket containing water. The blade and handle are made of a solid piece of metal, which obviates the dangers of sepsis peculiar to the complicated Simon instrument. The first stage in all these vaginal operations is to dilate, curette, and pack the uterus for the purpose of securing a healthy endometrium. Now that this has been done, I make a semicircular incision anterior to the cervix, as in the operation for hysterectomy. The bladder is then dissected from the uterus up to the fold of peritoneum, where it is reflected from the bladder on to the uterus. In dissecting the bladder from the uterus, I find it is easier to get into the line of cleavage by beginning the dissection at either side of the median line, where the bladder is readily peeled off by the handle of the scalpel. Directly in the median line is the attachment of the utero-vesical ligament, which must be cut in order to sever its attachment to the uterus. When this has been cut the bladder can be scraped free of the uterus with the handle of the scalpel, or can be pushed off by the finger. I prefer the latter method, after the utero-vesical ligament has been severed, making firm pressure with the ball of the finger. The fold of bladder at the point of attachment can be felt to yield under digital pressure, and this process is continued until the peritoneum is reached. My rule in this little procedure is never to allow my finger to advance unless I can feel the

tissue yielding and receding before it, constantly increasing the pressure of the finger against the cervix until that sensation is obtained. I now thrust my two index-fingers through the opening in the peritoneum and tear that membrane on either side at its junction with the uterus, to enlarge the field of view and of approach. I now grasp the severed edge of the vagina on either side of the middle point of the transverse incision with two artery clamps and drag down upon them until the vaginal wall is on the stretch. Beginning at the neck of the bladder, I now make a longitudinal incision the entire length of the anterior vaginal wall, going through the vaginal mucous membrane and sheath. With the handle of the scalpel I now dissect the bladder from the vagina for the distance of an inch on either side of this longitudinal incision. This makes a good free opening, as you see, into the pelvic cavity, and through this I propose to deliver into the vagina the fundus of the uterus, and after it the appendages, first of one side and then of the other. The vaginal retractor is now removed, and, dragging down firmly on the traction forceps, which still grasps the cervix, I pass my index-finger along the anterior wall of the uterus and hook it over the fundus. I find that the uterus is firmly bound to the rectum, but by a little manipulation the adhesions are severed, and I pass my finger on to the appendages of the left side. Here I find the tube and ovary bound together and firmly attached to the surrounding structures. Exploring now with my finger the right side of the pelvis, adhesions of cobweb consistency are discovered about the appendages, and they are easily broken up. The fundus and the appendages of the right side now being free, I hook my index-finger over the free border of the broad ligament near the right horn of the uterus and grasp between my thumb and finger the firm tissue of the round ligament. Dragging the horn of the uterus forward and pushing the cervix by means of the traction forceps back into the hollow of the sacrum, I am able to slip the bladder over the fundus, and here we have the uterus lying in the vagina, the fundus at the vulva. Without difficulty I now drag down the ovary and tube of the right side, and you can see that they are in a fairly healthy condition. By throwing a stream of salt solution upon the fimbriæ they float readily, and I have no difficulty in passing a fine silver probe into the Fallopian tube. The delicate bands of tissue you see adhering to the ovary are simply the remains of the cobweb adhesions which I have destroyed.

I replace the ovary and tube of the right side, and, dragging down upon the left broad ligament, I am able more readily to deal with the adhesions about the appendages of that side, which are now destroyed, and I am also able to deliver into the vagina, as you see, the ovary and tube, which are rolled up together in a somewhat confused ball of tissue. I separate the tube from the ovary without much difficulty, and we now see that the infundibulum of the tube is greatly enlarged; the walls are thickened, and a constriction at this point, about two inches from the uterus, marks the site of a stricture in the tube.

Here is an indication for conservative work, with the hope of preserving the functions both of the ovary and of the tube. Before severing the tube I quilt off the mesosalpinx with fine catgut from its free border up to the point of amputation. This will control the hemorrhage, and I now cut the mesosalpinx between the line of sutures and the tube up to the point of amputation, and also sever the tube. In order to maintain the patulous end of the tube at the point of amputation, and to prevent adhesions, the stump is treated in a manner analogous to that of the foreskin in the operation of circumcision. Inserting the sharp-pointed blade of the scissors into the canal of the tube for the distance of a half-inch, I slit up the stump on its posterior aspect; with fine catgut the mucous membrane lining the tube is now whipped by running stitches on to the peritoneum covering the tube, around the entire circumference of the incision. The ovary, you observe, is large, and at least one-half of it has been destroyed by cystic degeneration. I cut out this portion of the ovary opposite the hilum by incisions in its long axis, as you would slice a melon. This reduces the size of the ovary fully one-half, and the edges of the incision are brought together with a Lembert suture.

Before dismissing this part of the procedure I fasten the stump of the tube with a suture near the ovary. It now remains to shorten the round ligaments in order to retain the uterus in its normal position.

Retracting the vaginal wall upon the left side, I follow out the round ligament an inch and a half or two inches from the uterus and grasp it with an artery clamp. Making traction upon the clamp, I form a loop of ligament and, with a strong round-pointed but small needle, pass a silk suture through both segments of the round liga-

ment at the base of the loop; by tying this suture I shorten the ligament the entire length of the loop. To take the strain off this suture I pass another midway between it and the forceps, and now a third through the tip of the loop and the base of the round ligament where it springs from the uterus, and by tightening it fasten the loop of ligament just below and anterior to the horn of the uterus. This completes the operation upon that side, and, to reduce the strain upon these sutures to a minimum, I now rotate the uterus upon its long axis and bring the right horn of the uterus into more direct view.

The same method is repeated in shortening the round ligament on this side, and all that now remains is to let the fundus slip back into the pelvis, adjust the bladder to its normal position, and sew up the longitudinal incision in the vagina with a glover's stitch of catgut. A moderate amount of gauze is packed in the vagina, a self-retaining catheter is inserted into the bladder, and the patient is ready for bed. Upon inserting the catheter I am always careful to test for the presence of urine, so that I may know the bladder has not been wounded. That is an accident, however, which has never happened to me in these operations.

There is an important complication of chronic retroversion that is almost universally present in cases of congenital retrodisplacement, which requires serious consideration in deciding which operation shall be done to retain the uterus in its normal position. I refer to the short anterior vaginal wall or utero-vesical ligament. Unless the cervix is allowed to swing back freely into the hollow of the sacrum so that the intra-abdominal pressure is distributed naturally upon the uterus, there is no rational operation that will retain it permanently in its normal position. If an Alexander operation be done, the round ligaments are made to sustain the uterus, which hangs like a pendulum directly behind the symphysis, the anterior vaginal wall dragging the cervix forward to the same degree that the round ligaments drag the fundus.

The natural function of the round ligaments is not to sustain the weight of the uterus, but rather to maintain the proper position and limit the excursions of the fundus. The same condition obtains in these cases when the fundus is stitched to the abdominal wall, and whichever operation is done, the sustaining tissues sooner or later become stretched and the original condition is reproduced. This

result is especially common in cases of procidentia in which either hysterorrhaphy or the Alexander operation has been performed. In the operation that I have just done the vesico-uterine ligament is severed at its junction with the uterus, and the cervix is thereby allowed to swing back into the hollow of the sacrum as the fundus is drawn forward by the shortening of the round ligaments, and the normal position of the uterus is thus secured. The severed end of the vesico-uterine ligament reattaches itself to the anterior wall of the uterus at a point higher up than its original insertion, and no longer tends to drag the cervix forward. This is an essential factor in the success of the operation through the anterior vaginal fornix.

In the second case, which is now ready for operation, there is also retroversion with adhesions, but I can feel in the posterior wall of the uterus two hard protuberances about as large as olives which I take to be fibroid tumors. On either side of the fundus as it lies in Douglas's pouch I also detect a small, hard, irregular, movable body. In an examination without the anæsthetic I mistook these for the ovaries, but now that the patient is well relaxed, I can detect the ovaries lying to the outer side of these hard masses, and I therefore infer that the latter are pedunculated fibroid tumors. I am led to this conclusion from the presence of unmistakable fibroids in the wall of the uterus itself.

I shall operate through the anterior vaginal fornix as in the previous case. Having already explained all the steps in the technique, I need spend no time in describing the operation up to the point at which I enter the peritoneal cavity.

Passing my finger over the fundus, the adhesions yield readily to pressure, and I think I shall have no difficulty in delivering the fundus with the tumors into the vagina. I made a rather freer incision than usual to permit of this, and now you can see that those movable masses are really pedunculated fibroid tumors; the pedicle of the left one is so attenuated that the probabilities are that ere long it would have entirely separated from the uterus and have become a wandering tumor. Such tumors are not uncommon; sometimes they remain free in the abdominal cavity, and in some cases they reattach themselves to the tissue with which they lie most directly in contact.

With a slight twist and a firm pinch of my thumb and finger I separate this little tumor, which you see is about the size of a walnut.

The pedicle of the corresponding tumor of the other side I grasp with the forceps and cut away. As there is no hemorrhage from these pedicles, I will leave them until we shall have removed the tumors in the wall of the uterus; and now that we have the uterus in full view, we find that there are not only two, but four small fibroids here in the posterior wall. Two incisions will probably enable us to get them all out. You notice that I am obliged to cut through muscular tissue to reach them, and we must regard them, therefore, not as subperitoneal but as mural,—that is, imbedded in the wall itself.

Upon opening the capsule, you notice they shell out like peas from a pod. Now that I have made the second incision, I find that there is still another and smaller tumor present, and two more in the upper angle, so that we have three to remove through this incision, making in all seven tumors, five mural and two subperitoneal or pedunculated.

The free oozing that is apparent will readily be controlled by the sutures, which, as you see, I apply after the manner of the Lembert suture. The second incision, which is the larger of the two, I will suture with a buried catgut suture through the muscular structures and a continuous Lembert suture for the peritoneum. This patient is forty-three years of age, has not been pregnant for eight years, although she has borne three children, and has been suffering from excessive and too frequent menstruation.

I complete this operation by shortening the round ligaments, as in the previous case, and by closing the longitudinal incision in the vagina with a glover's stitch of catgut as before. Some gauze in the vagina and the insertion of a self-retaining catheter complete the operation.

Diseases of the Eye and Ear

DESCRIPTION OF A NEW METHOD FOR THE IMPLANTATION OF GLASS BALLS INTO TENON'S CAPSULE.[1]

PORTION OF A LECTURE DELIVERED BEFORE A CLASS OF PRIVATE STUDENTS.

BY CHARLES A. OLIVER, A.M., M.D.,

Attending Surgeon to Wills Eye Hospital; Ophthalmic Surgeon to the Philadelphia Hospital, Philadelphia.

GENTLEMEN,—A fair trial with the former methods of implantation of glass balls into Tenon's capsule has induced me to endeavor to devise a plan to obtain a more smoothly surfaced and a better rotating stump upon which to place the shell of a properly shaped artificial eye. After several attempts, I found that by separating and cleanly resecting the tendinous extremities of the four rectus muscles, and neatly suturing the associated parts together by a modification of the so-called corneal stitch of Kalt, I could obtain an overlying conjunctival surface which was free from nodulations and irregular cicatrices, and one that was devoid of any gross attachments to the underlying tissues.

At first my results were no better than with the other plans, but by repetition, selection of suitable cases, rigid asepsis, avoidance of useless and meddlesome detail, freedom from handling of structures that were intended for union, and enforcement of most careful

[1] The lecturer first gave a brief account of the indications for the performance of the operation itself, and described at length the details of the method employed by Mr. Frost, of London, with whom implantation of glass globes into Tenon's capsule originated. He then explained how Frost's plan of procedure had been modified by Mr. Lang, of the same city. This was followed by a description of the later independently devised plan of Dr. Morton, of Minneapolis, and the modifications that have been suggested and practised by Mr. Barrett, of Melbourne.

after-treatment, I have been able to bring the procedure to an almost uniformly successful termination (ninety-eight per cent.), and reduce the time of the healing process to a fewer days' duration than that consumed by an ordinary simple uncomplicated enucleation.

The procedure, which was first explained to the Section on Ophthalmology of the College of Physicians of Philadelphia at its April, 1899, meeting, and described at length in the May 27, 1899, number of the *Philadelphia Medical Journal*, is as follows:

"The conjunctiva around the entire corneal limbus is freed from the globe and then dissected sufficiently far back to expose the tendons of the four rectus muscles. The tendinous extremities of the muscles are made ready for separation from the globe. A half-curved needle with its point directed towards the corneal border, and holding a long piece of catgut thread, is carried directly through the belly of the internal or the external rectus muscle, and brought out of the tendon of the muscle just behind the remaining attachment to the globe. The muscle thus secured is cut loose from the globe just as in ordinary tenotomy. The catgut thread is drawn through as far as practicable, and a sufficient length of the strand of gut is left untouched to form a loop broad enough to admit of free manipulation between it and the eyeball. The needle is carried over to the opposite side of the cornea, and, with its point directed away from the cornea, is made to transfix the tendinous belly of the other lateral muscle, which is secured and freed from its connection with the eyeball.

"The vertically placed muscles are dealt with in a similar manner.

"The four rectus muscles are thus freed from their tendinous attachments to the globe, and each pair of muscles is secured in a loose sling that can be tied the moment that this becomes necessary.

"Working in between the broad loops of catgut attached to the ends of the muscles, which are held apart by an assistant, the eyeball is enucleated, with as much of the optic nerve as may be desired, without any difficulty.

"The cavity previously occupied by the globe is thoroughly cleansed and a water-tight glass ball of about three-fourths the size of the normal globe is dropped into place.

"The ends of the lateral rectus muscles which are held by the

lower and the first-placed catgut thread are neatly trimmed and sutured together. The same is done with the ends of the two vertical rectus muscles. The circular opening made by the cut edges of the overlying conjunctiva is lengthened into a lozenge by a couple of horizontal snips, and is carefully brought into linear apposition by a series of silk threads.

"The operative field is covered by a gauze protective bandage upon which iced compresses are placed."

The first of the following sketches shows the appearance of the parts just previous to the insertion of the vertically placed catgut ligature into the still attached tendon of the inferior rectus muscle. The speculum is in position. The conjunctival membrane has been freed from the globe at the corneal limbus. Both of the lateral

FIG. 1.

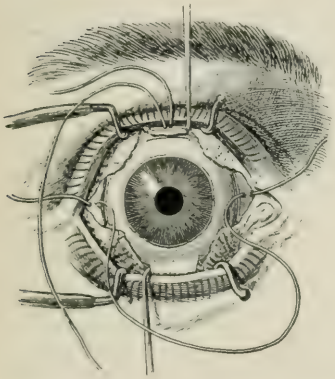
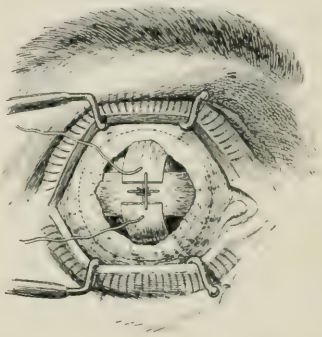


FIG. 2.



muscles have had the horizontally situated catgut suture placed in position and their tendinous insertions dissected loose from the eyeball. The broad loop connecting the ends of the two lateral sutures, through which the eyeball is to pass at the time of enucleation, is well shown.

The long loose suture in the free extremity of the superior rectus muscle, with an underlying hook in order to exhibit the surface of the resected end of the muscle, is ready to be inserted into the tendon of the inferior rectus muscle, which is being held ready in position by the lower hook.

In the second sketch it will be noticed that the eyeball has been removed and replaced by the glass ball. The resected ends of the two lateral rectus muscles have been brought together in front of the

glass ball, and sutured into position in such a manner that their free extremities approximate without the intervention of any foreign substance. The appearance of the stitch is very well shown in the sketch.

The overlying vertical muscles have been caught in the vertically placed suture, and are ready to be approximated and tied over the already sutured lateral muscular bands; the two sets of sutures, being independent of one another and well separated, permit the production of an absolutely flat and firm double barrier for the prevention of the escape of the underlying glass globe situated within the capsule of Tenon.

The third sketch shows both the lateral and the vertical muscles sutured into position over the glass globe. The conjunctival opening has been lengthened laterally. All of the superficial silk su-

Fig. 3.

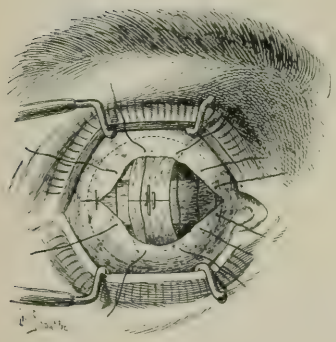
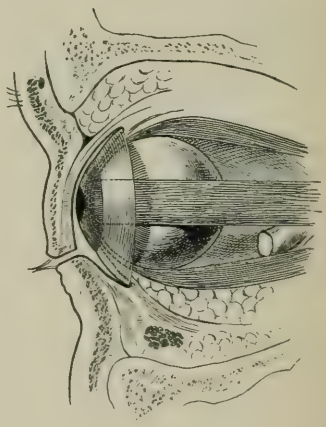


Fig. 4.



tures have been placed in position, and the outer one has been tied; the operation being practically finished as soon as the conjunctival wound has been brought together throughout its length by the remaining five threads.

The fourth sketch gives a lateral sectional view of the lids, the conjunctival sac, and the bony portion of the anterior part of the orbit. Into the orbital cavity have been placed the glass globe, the rectus muscles, a portion of the capsule of Tenon, some fat, and the stump of the optic nerve. The oblique muscles, the levator of the upper lid, the greater part of Tenon's capsule, as well as the

nerves, the vessels, and the ligamental tissues, have all been purposely omitted in order to avoid confusion during the study of the sketch.

In the conjunctival sac a properly shaped and well-placed artificial eye can be seen resting on the conjunctiva, which in turn is smoothly and evenly supported by the underlying muscular structures and glass globe, thus illustrating graphically, as it were, how well adapted the parts have been made for the free movement of a well-fitting shell.

SPRING CONJUNCTIVITIS, OR VERNAL CATARRH; ULCERATION OF THE CORNEA; MARGINAL KERATITIS; GLAUCOMA EXCAVATION; GLAUCOMA.

CLINICAL LECTURE DELIVERED BEFORE THE MISSISSIPPI VALLEY MEDICAL ASSOCIATION.

BY F. C. HOTZ, M.D.,

Professor of Ophthalmology and Otology in Rush Medical College, Chicago, etc.

SPRING CONJUNCTIVITIS, OR VERNAL CATARRH.

GENTLEMEN,—The first case I present to you is a boy who every summer for the last three years has been troubled with irritation and an uncomfortable sensation in his eyes. They become inflamed, and there are watery secretion and great sensitiveness to light. In autumn his eyes get better, and in winter they seem normal. This recurrence of the disease every summer points to the presence of a very interesting and peculiar disease of the conjunctiva, the so-called "spring catarrh." It was given this name by Professor Horner, of Zurich, because in Switzerland and South Germany, where Dr. Horner practised, the warm season begins early in spring, and not, as in this country, in June or July; here it should be called "summer catarrh," for there is a close relation between this affection and warm weather. It comes with warm weather and goes with warm weather; and even during the summer, if such a patient be under observation, a marked variation or oscillation of the symptoms is noticed in strict relation to the weather. When cold winds prevail in summer and the temperature is rather low, the eyes will improve, and you may flatter yourself with the great success of your treatment; but as soon as the wind veers towards the south and the temperature rises, your enthusiasm will cool; for the inflammation in the eyes will rise with the temperature, so that you will have to revise the conclusions drawn from the apparent improvement as the result of treatment; and so it will go up and down, up and down, until cold weather sets in.

To-day, after this unusually cool spell, which is more like that of November than September, this boy's eyes are in much better condition than they were a few weeks ago, when we had unseasonably hot weather.

The disease shows such characteristic features that, having once seen it, you will recognize the affection immediately. It begins in the tarsal conjunctiva, causing redness, thickening, and enlargement of the papillæ, so that the conjunctival surface of the lid appears roughened and granular, and might be mistaken for granular conjunctivitis. When the tarsal surface is inspected, the conjunctiva is not bright red, as in catarrhal or granular conjunctivitis, but the whole surface appears as if it were coated with a layer of cream or milk, or of white paint. The lower lids in this case show very little change; they appear almost normal; but when I expose the upper lids you perceive a peculiar condition. The papillæ are enlarged, especially along the upper borders; the surface is slightly granular, but not congested; and there is a peculiar milky, whitish haze all over it.

While I am demonstrating this conjunctiva, I wish to show you an easy way of everting the eyelid, so that you can examine the whole conjunctiva. As the lid is everted you can see only the portion of the conjunctiva that covers the eyelid and the conjunctival portion that covers the eyeball. Between these two parts there is the so-called retrotarsal fold, or the fornix which unites the ocular and the tarsal conjunctivæ. In the ordinary eversion of the lid, as I have done it, to demonstrate the condition of the tarsal conjunctiva, you cannot see the retrotarsal fold, which might be seriously diseased and you not know it, because it does not show itself unless it is so swollen that when you turn the lid it springs out spontaneously on account of its size. In order to inspect this fold it is usually advised to lift the upper border of the everted lid away from the eyeball with a probe, a spatula, or a forceps, so that you can look under the lid border; or, you may be directed to grasp the upper border of the everted lid with a forceps and draw it away from the eye. These procedures are more or less painful unless you cocainize the eye; but they are unnecessary, as I will convince you directly. It is easy to bring the retrotarsal fold into view without inflicting the slightest pain on the patient. All that is necessary to do is, after turning the lid over in the

ordinary way, to hold with your finger the free border of the everted lid firmly against the upper margin of the orbit, and to place a finger of your other hand on the lower lid, push it over the eyeball, and gently press the ball backward. You see when I do so the fold comes out at once; it is swollen; and when I discontinue the pressure it recedes again. This manipulation does not hurt the boy at all. This procedure is little known; but ever since I have discovered and practised it I have found it invaluable in the thorough examination of the conjunctiva of the upper lid. It is simple and absolutely painless; it can be executed without any instrument, and will often reveal conditions of the retrotarsal fold that are not suspected.

In this case the affection has so far been limited to the lid conjunctiva. In another class of cases this vernal, or spring, catarrh causes an infiltration around the cornea in the sclerocorneal border or limbus of the conjunctiva.

With regard to the prognosis, we cannot say positively how soon a patient will recover. In many cases the attacks recur for a number of years and then cease. In other cases the attacks occur indefinitely every spring or summer, and every year the condition becomes worse, so that eventually the whole lid conjunctiva assumes the thick, rough, infiltrated condition called granular conjunctivitis.

In regard to the treatment, we must confess that we have here a condition that we cannot control. So far, all remedies which have been tried have not proved a positive success, so that in the chronic forms of the disease, with thickened folds and enlarged papillæ, the scraping process,—so-called curettage,—with corrosive sublimate, has been resorted to; and some assert that they have completely eradicated the trouble by this treatment. The conjunctiva is scrubbed with a rough bristle brush and corrosive sublimate, or scraped with a sharp curette and rubbed with corrosive sublimate, one to five thousand. In the milder cases oxide of mercury ointment and all the eye lotions imaginable have been tried. Last year, in this case, we tried protargol, a new silver salt, which makes a deep coffee-brown solution; and I would advise the use of it again on account of the succulence of the retrotarsal folds. Protargol is a substitute for silver nitrate, and is not painful, not irritating like silver nitrate, even when used in twenty per cent. and stronger solutions. I now always use at least a twenty per cent. solution,

which is brushed over the conjunction of the lid and fornix with a camel's-hair pencil once every day.

ULCERATION OF THE CORNEA.

The next two cases I have to present are excellent examples of ulceration of the cornea. This boy shows in the centre of the right eye a grayish, dull-looking spot, accompanied by injection of the eyeball, the so-called pericorneal injection. He has suffered from an affection of the cornea for some time, and when you examine the eye closely, you see, especially below and in contact with this dull, grayish opacity, a faint cloud over which the epithelium is bright and smooth, showing that the opacity is old. In fact, it is a slight opacity left from former ulceration. The dulness in the centre of the opacity shows that it is recent; that there is active ulceration still going on. This ulcerative process is superficial, limited to the epithelial layers of the cornea, and is the result of so-called phlyctenular keratitis. The pupil is dilated by atropine, which constitutes the main feature of the treatment in cases of superficial ulcers or superficial keratitis.

I will now show you a different form of ulceration of the cornea in this old man, whose left eye has been inflamed off and on for the last three weeks. He has been in the hospital a week since Saturday, under the care of Dr. Montgomery, through whose kindness I am able to present him here. He has considerable pain in the eye. The pupil is fully dilated by atropine. The eyeball shows very much injection. The whole sclera is covered by a characteristic pink injection of the ciliary vessels, forming a broad, roseate band around the cornea. The cornea, you see, has a dull appearance; the iris and pupil, seen through the cornea, do not show so bright, and the color is not so clear and distinct as in the other eye. The point to which I call your attention particularly is a ring half-way between the margin and centre of the cornea, like a circular groove cut with a needle. This groove appears in parts gray and in other parts almost transparent. If you look closely, you see that the ring is irregular; that the margin of the groove towards the centre is slightly elevated and gray, which is an expression of infiltration and an indication that the process is progressive. In this case the process started in the upper half of the cornea, and when Dr. Montgomery saw the case on admission to the

hospital it had formed a half-ring in this part of the cornea. It has since gone on to a complete ring by involving the lower half. This is a so-called ring ulcer, a suppurative process due to infection, which is one of the most serious affections of the eyeball. The patient's suffering is less than it has been. His eye generally feels better while under treatment. Dr. Montgomery has directed treatment of the general health, as the patient was run down; his general condition is improving, which is an additional help towards improving the resisting power of the tissues. I said it is a suppurative process, one form of suppurative ulceration in the cornea, of which there are quite a number of varieties, all due to infection, and bacteriological examinations always prove the presence of pus cocci.

I desire to make a few remarks in regard to the treatment. Since it has been established that these suppurative processes in the cornea, like any other suppurative process, are due to the presence of bacteria, some physicians seem to think that bacteria-killing is the sole indication, and that instillations of carbolic acid, sublimate, formalin, etc., are all that is required in the treatment of suppurative inflammations of the cornea. Now, to get rid of the cause of the disease is certainly a most rational procedure. But I dare say that the instillations of these germicidal solutions are ineffective. Because a solution of corrosive sublimate, one to five thousand, kills bacteria in the test-tube in a certain length of time does not justify us in assuming that a like solution dropped into the conjunctival sac of the eye will accomplish the same thing. It cannot do it because it does not remain in prolonged contact with the germs as in the test-tube. If any of you happen to remember that about ten years ago I advocated the use of corrosive sublimate solution, one to five thousand, in the treatment of suppurative ulcerations of the cornea, you might think that I now consider my advocacy of corrosive sublimate at that time as an error; but this is not so, as you will understand when I describe my method of using the sublimate. The only way to insure the action of corrosive sublimate, or any such antiseptic lotion, upon the bacteria present in an ulcerated cornea is to have the patient tilt his head away back, so that the eyeball is the lowest part of the face, like this (illustrating); then, while the nurse is holding the eyelids well apart, fill the whole space, with the eyeball as the lowest part of the

cavity, with the solution, so that the cornea is submerged in the corrosive sublimate solution, and keep it there for at least ten minutes. Treated in this manner, I have seen suppuration of the cornea arrested quickly and progressive ulcers heal in a comparatively short time; and the accumulation of pus in the anterior chamber, —so-called hypopyon,—so frequently found in connection with these ulcers, sometimes disappeared in twenty-four hours. This is a different way of using sublimate from simply dropping it upon the eyeball once, twice, or three times a day.

We have, however, a much more reliable and efficient application to arrest the suppurative process in the cornea, and that is heat, applied in the form of a red-hot iron, a steel probe heated in a spirit-lamp, or a platinum loop heated by electricity. The electro-cautery instrument is the most convenient, because you can apply the platinum point near the margin of the ulcer cold, and then by pressing the button the loop becomes heated at once and cauterizes the infiltrated tissue. If you have no such appliance, any small steel probe, or even a common sewing-needle, fastened with its point in a wooden toothpick, forms a good enough appliance for extemporaneous work. The only trouble is, you have to heat it in the spirit-lamp away from the eye, and by the time you transfer it to the eye the heat is partially lost, and you have to be very quick in touching the ulcer. This cauterization by heated metal passed gently over the infiltrated margin of the ulcer is the surest way of arresting suppuration. It is surprising how rapidly healing takes place after such cauterizations, with a smoother and less opaque cornea than after any other treatment, so that I prefer its application in all cases as the principal treatment.

But, besides the cauterization for the purpose of destroying live germs in the tissues, we must never forget to put the eye at rest by the use of a mydriatic, to stop the action of the pupil, to rest the ciliary muscle. Besides, we must protect the eye against dust and impurities; and if the friction of the winking lid over the ulcerated cornea is painful or irritating, we must bandage the affected eye, and in very serious cases it may become necessary to bandage both eyes, to insure as much rest and immobility of the eyeballs as we can obtain. The building up of the general constitution of the patient is another feature in the treatment.

MARGINAL KERATITIS.

This case is an example of another form of corneal affection limited to the margin of the cornea. The case does not show the features so well as it did a few days ago. To-day there is a little red patch at the lower nasal margin of the cornea, and on close inspection you find there a little irregularity, as if fine particles of dust had been deposited. The right eye shows two patches of injection, one on the temporal and one on the nasal side of the cornea, and in the corresponding portion of the margin of the cornea you notice the same condition as you see in the left. This case is called marginal keratitis,—that is, a phlyctenular inflammation with an eruption of small nodules in the conjunctival limbus. Properly speaking, this case belongs to the class of phlyctenular conjunctivitis. It is the conjunctiva that is affected here chiefly, and the injection is all conjunctival.

From the clinical stand-point, we must make a distinction between the phlyctenular eruptions of the conjunctiva and the phlyctenules in the cornea. In the conjunctival affection, the application of the yellow oxide of mercury ointment, about two per cent., a small bit of it placed into the conjunctival sac and gently rubbed and spread over the eye, will invariably cause absorption and disappearance of the phlyctenules of the conjunctiva; while on the phlyctenules which appear in the transparent cornea the ointment acts as an irritant and causes a good deal of pain. It, therefore, should not be used during the period of acute inflammation. In the corneal phlyctenules atropine is the mainstay of our treatment, to rest the eye until the inflammatory stage has passed away. The eyes should also be protected against bright light, smoke, and dust by properly tinted glasses; but pure air should not be excluded from the eyes, and bandages, therefore, should not be allowed except for the temporary applications of moist compresses, which are very soothing and efficient in relieving lid spasms.

GLAUCOMA EXCAVATION.

This woman is sixty-two years of age. She comes to us with the statement that for the past two years she has had occasional obscuration of sight; at times it was practically lost. The sight then returned and remained good until another attack occurred. When the clouding of sight occurred she had a feeling of fulness

or of pressure, but no decided headache. Another symptom is that in looking at a lamp or gaslight she sees a prismatic play of colors around it. She does not suffer from marked pain, even during the severest attacks.

The examination of her eyes, as far as you can inspect them with the naked eye, shows a normal condition. The cornea is clear, smooth, and transparent. The iris is perfect in color, and the anterior chamber has the normal depth. The pupils have the small diameter seen in old people. With the ophthalmoscope you will find the lens clear and transparent; the vitreous is also clear. You have no difficulty in obtaining a good view of the fundus, and this will at once reveal to you the nature of her trouble. You will observe that the optic disk shows the characteristic appearance of the so-called glaucoma excavation.

In the normal optic disk you can trace with your eye the retinal vessels from the point of emergence uninterruptedly over the margin of the disk on to the retina. In glaucoma excavation the blood-vessel, after passing across the floor of the disk, has to ascend the steep side of the excavation to appear on the retina. The result is that we see a blood-vessel crossing from the centre of the disk to the margin, but we cannot trace it over the margin; and we find another blood-vessel starting at the margin, but not continuous with the one we saw on the disk. We do not see the ascending part of the blood-vessel, and it appears as if the blood-vessel has been shifted to one side; and by moving your head while keeping the optic disk in view, you see the shifting counter-movements of these two blood-vessels in the same way as you see two objects situated at different distances before you apparently move in opposite directions when you move your head to one side: the nearer object moves in the opposite direction and the farther object moves in the same direction as you do.

Vision in the right eye is normal; vision in the left eye is only 15/40; the field of vision shows a marked constriction.

The condition of glaucoma is due to increased tension. The increase of intra-ocular tension is the fundamental condition of glaucoma. We can ascertain the tension by palpation. Having the patient close the eye, with one finger gently exert a slight pressure upon the eyeball, while with the other finger determine how much resistance the eyeball offers to the pressure. The manipula-

tion is exactly the same as you use for detecting the sign of so-called fluctuation. If the tension is increased, the eyeball offers greater resistance to the finger than under normal conditions; it appears fuller or harder. In glaucoma with a very high degree of intraocular tension, the eyeball will give a feeling to the finger as if it presses upon wood or stone. In our case the tension to-day is apparently normal. This shows that the glaucoma storm comes on periodically, and that the tension decreases when the storm has passed; otherwise the sight would not be as good as we find it, after such a long time, and the field of vision would be more contracted also. But to-morrow we might find the tension more or less increased. That is just the way it varies in a certain class of cases of glaucoma, and it is this variation of tension which makes the disease so treacherous in these cases at the onset. Unless the patient is carefully and repeatedly examined, you might easily make a mistake in your diagnosis. You might from the history suspect glaucoma; but finding the tension normal and no marked excavation of the optic disk, you might discuss the case as not glaucomatous. But a very close inspection may show the smaller blood-vessels just beginning to bend like the one in the picture I have shown you, and thus confirm your suspicion of glaucoma.

We know how important it is to take at once the proper measures in a case of glaucoma. No medicine, internally or locally applied, will arrest the progress of this disease and ward off the final doom of complete blindness. The only way by which glaucoma can be prevented from destroying the optic nerve is by operative procedures, chiefly a properly executed iridectomy. The importance of being sure of the diagnosis—making it in time before the optic nerve has suffered too much—cannot be overestimated. It is operative procedure that we shall have to suggest to this patient. The field of vision shows contraction, the optic nerve is excavated, and the glaucomatous attacks are characteristic. But I must add that in this case the curative effect of an iridectomy is not so absolutely certain as it is in well-developed acute or so-called inflammatory glaucoma.

GLAUCOMA.

Here is a case which illustrates the ravages of unchecked glaucoma. The lower temporal section of the field only is preserved in the left eye, though central or direct vision is still fair, 20/40.

If you examine such a case and find good vision, you might feel satisfied that there is nothing wrong with the optic nerve, and neglect to take the field; but if you map out the field with the perimeter, and find three-quarters of the field gone completely, you will become rather dubious about the future of the eye. Further examination shows that tension in this eye is decidedly increased, and the ophthalmoscope shows deep glaucoma excavation. When the field is reduced to that extent the prognosis is bad, although the central vision be still satisfactory. Even if an operation succeeds in controlling the tension, the degeneration of the optic nerve is likely to progress, and the extinction of all vision is the finale.

PRACTICAL REMARKS ON THE HYGIENE OF THE EYE.

CLINICAL LECTURE DELIVERED AT THE QUINZE-VINGTS HOSPITAL, PARIS.

BY E. VALUDE, M.D.,

Physician to the Clinique Nationale des Quinze-Vingts.

Gentlemen,—The eyes of human beings require attention from the moment they are born; by this I mean exactly what I have said, as it has been shown, in the statistics published by Olshausen, that, in order to be efficacious, prophylaxis against ophthalmia neonatorum must begin even before the cord is tied.

The accoucheur's first care must be to preserve the infant's eyes from the ophthalmia to which they are exposed. The measure usually adopted is to instil a few drops of a two per cent. solution of silver nitrate between the child's lids. Some accoucheurs are satisfied with simply washing the eyes with a solution of boric acid; corrosive sublimate must be avoided altogether, as the conjunctiva at birth will not tolerate it in any dilution. Instead of the silver nitrate, M. Pinard uses lemon-juice, an article that can always be procured in every family. For my part, on account of the irritation caused by lemon-juice and silver nitrate, I have avoided their use and have had recourse to powdered iodoform, which has been successfully employed for eight years in some of the obstetrical wards of Paris. M. Porak has recently experimented, in this connection, with a substance that is highly thought of at present as an antiseptic, —formol,—and my own opinion is very favorable to the idea.

Although formol has not the same microbicidal potency as sublimate, it is far superior to it as a means for disinfecting a region. That is to say, although sublimate applied to the conjunctiva is more efficacious than formol in destroying the microbes that may be present, it does not prevent the subsequent development of the spores that may have escaped its immediate effect. Formol, on the other

hand, prevents altogether and for a long period all microbian growth. This peculiarity of formol is not generally known, and its value in preventing ocular infection will readily be understood. I always use formol in preparing a patient for an operation for cataract.

Formol can, then, be used in a one per thousand solution as a means for preventing ophthalmia neonatorum; a few drops of such a solution should be introduced between the child's lids.

During early infancy a child's eyes have only to be kept clean, and this can satisfactorily be done with a saturated solution of boric acid used warm.

The question of how the cradle should be placed as regards the light and to prevent strabismus is a pure matter of superstition. All that is true is that for several months the equilibrium of the eyes, as regards binocular vision, is not established in children; so it is perhaps desirable that it should not be solicited by a bright light coming always from the same side.

During childhood the hygiene of the eyes is again solely a matter of cleanliness; but at this period it is not enough to rely only on the boric solution; parents must pay attention to the condition of the child's hands, clothes, toys, and to the children chosen as playmates. Among the ocular disorders of this time of life phlyctenular ophthalmia occurs in as high a proportion as ninety per cent. or more, and is often associated with impetigo of the face, scalp, or nasal fossæ. Inoculation of the eyes may be brought about by dirtiness of the child's fingers or by lack of care of the diseased regions. As a means of avoiding this form of ophthalmia, the children must be kept very clean, with short hair, and nails carefully trimmed; and, whenever possible, they should have wholesome food and be placed in proper surroundings, though this is often impossible in the class of society that furnishes the greater part of this contingent of patients.

Of children's toys, it is well to guard against all that have points or sharp edges, particularly the former. The number of children is quite large who injure their eyes with scissors or pens, and this mishap is often followed by serious infection or traumatic cataract.

In addition to phlyctenular ophthalmia and accidents, children are liable to contract a form of conjunctivitis, called catarrhal, due to a special bacillus (Weeks). This complaint is caught from other

children, and often spreads rapidly through asylums and primary schools. Every child suffering from this form of conjunctivitis should be isolated from the others until recovery is assured.

When children are old enough to go to school, not only do the foregoing considerations have to be borne in mind, but the hygiene of vision has to be added to them. This problem should be regarded from two points of view,—the general measures applicable to all children, especially the question of light; and the special rules applicable to each case.

Thus, it is well that a child should not begin to read before the age of five, and six would be more preferable still. There is a tendency at present to begin children's education earlier and earlier on the pretence that they have now so much to learn; but in view of the slowness with which very young children acquire the elementary notions of reading, it would undoubtedly be better to wait until their power of attention is more fully developed, when they will learn the same amount in much less time. The question arises whether children are not really sent to school to restore quiet to the house! No prolonged effort of convergence should be permitted before the age of five or six, particularly when any tendency to myopia is present.

It would be very desirable to test the refraction of every child before it begins its studies. How many myopic and astigmatic children do we not see who have been allowed to assume defective positions that cannot be corrected, even when proper glasses are applied!

Strabismus could quite frequently be prevented if children who are known to have hypermetropic sight were supplied with glasses to relieve accommodation during the preparation of their lessons.

In daytime every child should be able to see the sky from its place for a space measuring not less than thirty centimetres (twelve inches), and light coming from both sides is best. At night each child should have a lamp furnished with a shade.

Children's desks and seats should fill the following five conditions: first, the seat should be close to the desk; secondly, the height of the desk such that the elbow rests on it when in its natural position; thirdly, the back of the chair at such a distance as to give support when writing; fourthly, there should be a foot-rest; fifthly, the top of the desk should be inclined at an angle of twelve degrees.

The distance from the eyes to the paper may vary between twenty-five and thirty-three centimetres (ten and thirteen inches).

During the early school years the children should be obliged to follow the precept formulated by George Sand,—writing straight, paper straight, body straight. Later they may be allowed to turn the paper to the left, to obtain greater speed in writing.

Books should be clearly printed; when lighted by a candle one metre off the print should be legible to good eyes at eighty centimetres (thirty-two inches). The details of maps should be legible at forty centimetres (sixteen inches).

Young children should not be kept at work more than an hour at a time; in primary schools this period can be extended to one and a half hours; but no child should be kept at continuous near work for more than two hours.

When a child is known to be near-sighted some of the foregoing regulations must be applied with special rigor. The light, for instance, must be the best obtainable, and the hours of study should be shorter than for those with normal eyes; but the most important point of all is to watch the distance at which near work is done. In no instance should the head be brought nearer than twenty centimetres (eight inches) to the book or paper. If the myopia is very marked, glasses should be given that permit vision beyond this limit; but when the myopia is less than six dioptres, reading at this distance can be accomplished without glasses. It is absolutely essential that the myope should never read at a shorter distance; each moment of work at closer range has a deleterious effect on the myopia, and the myopic patient who makes it an absolute rule never to read at a short distance may be sure that the use of his eyes will not then increase his refractive error.

With astigmatic or hypermetropic children it is different, as these conditions are not influenced by near work. In hypermetropia, the development of strabismus is to be feared from the undue convergence attending excessive efforts of accommodation. This can often be prevented by prescribing suitable convex glasses. If intermittent or alternating strabismus be already present, convex glasses should be worn constantly. Suitable cylindrical or sphero-cylindrical glasses should be constantly worn when astigmatism is present, not that we have in this case to fear excessive convergence, but be-

cause this disorder, when not corrected, occasions eye-strain and abnormal attitudes of the head and neck.

When the period of adult life is reached professional hygiene of the sight has to be considered. This we can only touch on in its main points, as the limits of our time will not permit us to discuss the requirements of each particular profession.

Here, as in all other cases, the question of illumination is the most important one. The various sources of light can be ranged as follows in the order of merit: incandescent electric burners; oil lamps; Auer burners; ordinary petroleum burners with round wicks; gas burners with chimneys; intensive petroleum burners.

It will be noticed that Motais, to whom this scale is due, thinks the electric light a good one, differing therein from many other authorities. The incandescent electric light is the one that is most like the light of the sun. When the eyes do not tolerate it, it is probably because it is improperly used. No one can look directly at the sun for any length of time; why, then, be surprised that it should tire the eyes to look at an electric light?

When the electric burners are arranged so as not to throw their light directly into the eyes, they constitute the best form of light. For ordinary use a sixteen-candle incandescent light with an opaque shade is perfection. The ordinary oil lamp is excellent, and the light from a petroleum burner, or from an Auer cone, also very good. The objectionable feature of lighting by gas is the heat it generates.

The same conditions govern the adjustment of the height of the table with reference to seat, position, and distance of the head, and the use of glasses in the case of grown persons as with children; convex glasses are, however, required by adults towards middle age to help out the accommodation, which becomes less adequate from year to year, irrespective of the state of refraction.

Mention must be made of the habit of reading in bed, to which so many are addicted, as it may do the eyes great injury, even when the sight is good, but particularly when myopia is present. Reading in bed is generally done with inadequate and badly placed light, and in a constrained position of the head and neck; both of these particulars have a most deplorable effect on sight. The same can be said of reading in carriages and on railway trains, when the vibration and motion together make reading very fatiguing.

Although I have just said that electric light closely resembles

the light of day, it is far better for the eyes' sake to work by daylight. Even when no special eyework or reading is being done, it is a bad plan to keep late hours, and society people, who often spend afternoons and evenings in intense artificial light, are subject to very stubborn attacks of blepharo-conjunctivitis and of papillary conjunctivitis, which crop up regularly every winter in a city population.

Natural light and free ventilation of dwellings are most important. Rooms with insufficient light and air, especially when they are damp, are a frequent cause of chronic papillary conjunctivitis.

Food has but little influence in ocular hygiene. We must, however, except the excessive use of alcohol and of tobacco, which may give rise to double optic neuritis, ending in complete atrophy and blindness.

The next question in order of interest is at what age presbyopia commences and glasses should be advised. There exists in this connection a deeply rooted prejudice that leads people to put off the use of glasses as long as possible, under the pretext that they weaken the sight. It is just the contrary that is true: sight is damaged by straining when glasses are necessary. At about the age of forty-two a person with good eyes would derive benefit from helping his accommodation out with glasses; at forty-five he should certainly take to them if he has not already done so; while at fifty, if he is still not using them, he is taxing his ciliary muscle to such a degree that it cannot fail to do him harm. It is true that once the use of glasses has been resorted to they can no longer be dispensed with; but it is equally certain that instead of weakening sight they do away with straining for accommodation.

In presbyopia convex glasses should be made stronger every three or four years; as a usual thing, at about the age of seventy sight remains stationary.

In advanced age there are no special rules of ocular hygiene. The lids, however, lack tone and are very liable to inflammation at this time of life, and antiseptic lotions become almost a necessity.

The rules laid down for lighting schools are also applicable to offices and counting-rooms.

It is a great advantage when factories, with their own motive power, can be lighted by electricity. The ideal system, and the one which gives the most satisfactory illumination, is that of Jaspard:

powerful burners are set in reversed conical reflectors placed so as to throw their light on the ceiling and walls, whence it is reflected as diffused light very similar to daylight.

The requirements of certain occupations necessitate work that is very fatiguing to the eyes or very irritating to them.

In the first case (type-setters, engravers, seamstresses, accountants), the important thing is to detect any error of refraction and correct it by suitable glasses.

In the second case, the eyes may be damaged by chemical action (fumes of sulphur or other irritating gases), by the various forms of dust (bakers, wool-combers), or by the presence of foreign bodies (metal-workers, grinders, hay-makers). A great many of these dangers can be avoided by wearing protective glasses, and antiseptic lotions will prevent ocular lesions from becoming infected and consequently serious. The gravity of injuries done to the eyeball by fragments of flying metal and other foreign bodies is due to secondary infection, usually resulting from lack of cleanliness.

THE EARLY OCULAR SYMPTOMS OF TABES DORSALIS, WITH A REPORT OF FOURTEEN CASES AND EIGHT CHARTS OF THE FIELD OF VISION.

BY LOUIS STRICKER, M.D.,

Of Cincinnati, Ohio.

Primarily, the symptoms of tabes are due to involvement either of the ganglia at the base of the brain or of the ganglia of the posterior roots of the cord. The pathological changes in the cord proper are secondary,—that is, the axons in the posterior columns of the cord die; the formation of the glia tissue is a subsequent process. The ganglion cells in the posterior horns of the gray cord may, however, be affected primarily in the same way as are the ganglia of the brain.

The retina is pushed outward from the brain in the early stage of fœtal development. The fibres of the optic nerve originate in the ganglion cells of the retina. These ganglion cells preside over the nutrition of the centripetal axis-cylinders, or sensory axons, which extend backward to the molecular layer of the cortex of the brain to the primary optic centres, where they break up into fibrils. Another set of centrifugal nerve-fibres originates in the brain, joins the optic nerve, and is distributed in the retina to the inner plexiform layers.

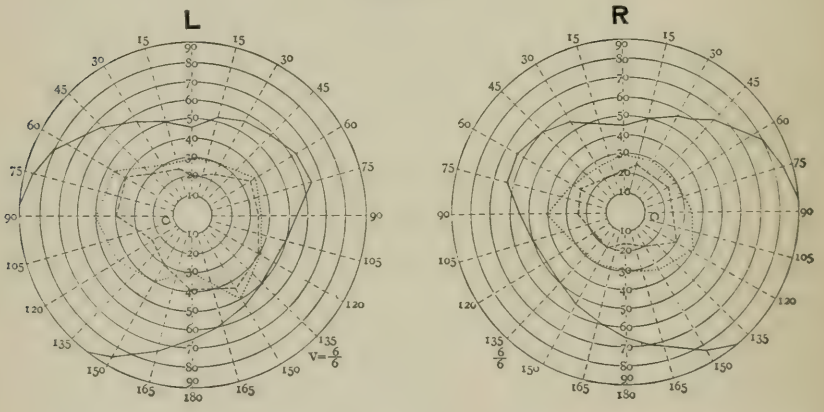
The fact that such widely separated portions of the cerebro-spinal axis may be primarily attacked in the course of the same disease indicates a similarity of cause in all. It seems rational to accept, as the causative factor, a toxic agent circulating in the blood, which by interference with the nutrition of the individual neurons causes death of their axons, and finally of the cell-bodies. Syphilis is the cause of this chronic toxæmia in about ninety per cent.—some authorities say one hundred per cent.—of the cases. This fact has not been established by deductive reasoning, but by direct inquiry and investigation in large numbers of cases as to the presence of syphilis in the individual, exposure to cold and violence being pre-

disposing factors which cause the disease to manifest itself first at points which have been previously weakened by excessive functional activity or by disease.

In a recent paper, Dana, of New York, says, "If there were no syphilis there would be no tabes." He believes that during the primary invasion of the disease the vitality of the nerve-cells is so affected that their period of life is shortened. Early senility of the system of neurons and premature old age are thus due in part to a congenital or acquired weakness and in part to toxic action.

Tabes is not an inflammatory disease. It is a primary atrophy or decay. Primary atrophy has long been recognized among diseases of the optic nerve, and is frequently associated with tabes.

Case I.



Tabes and spinal or cerebro-spinal syphilis present the same ocular symptoms early in their course; and should diplopia, ptosis, or paralysis of accommodation interfere materially with perfect vision, the individual soon seeks the aid of an oculist. In other cases, again, ocular symptoms may exist for a long time without making their presence manifest, and are frequently overlooked until more definite symptoms of spinal disease arrest attention.

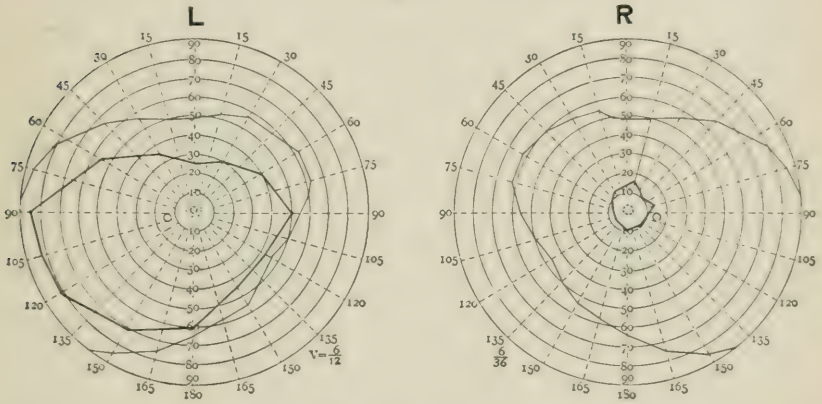
Statistics as to the frequency of special symptoms in tabes are of little value. Ocular symptoms are said to appear early in about fifteen per cent. of the cases. Some authorities place the ratio much higher. The oculist is prone to consider the percentage of cases with ocular symptoms high, since it is this class of cases that

comes under his observation, whereas the neurologist finds the spinal symptoms predominating in the larger proportion of his cases; these, as a rule, being most prominently developed when his aid is sought. The early ocular paralyses sometimes pass away, and are not present in the later stages of the disease.

The ocular symptoms belong to the early manifestations; some are fleeting, others permanent. In all the cases which have come under my observation in private practice I have failed but twice (Cases VI. and XIII.) to find combined with the ocular some of the prominent spinal symptoms.

Paralyses of the extrinsic ocular muscles, at times but partial or transient, make further investigations imperative; these often disclose symptoms which, though less annoying, are nevertheless

Case II.

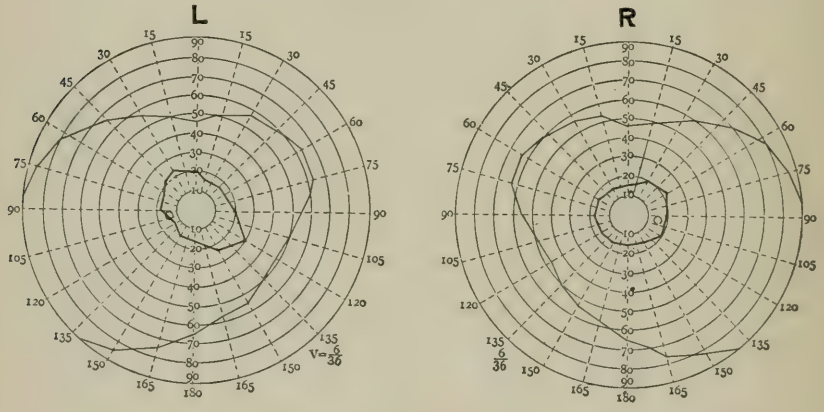


of great diagnostic and prognostic value, as, for example, inequality of the pupils or spastic contraction of one or both of them, paralysis of the sphincter of the iris, associated at times with paralysis of accommodation; a combination of the last two conditions is looked upon as positive evidence of involvement of the ganglia in the floor of the third ventricle.

The oculomotor is the most frequently implicated of all the cranial nerves. Among the most constant phenomena of its involvement is contraction of the pupil during the act of accommodation, with absence of reaction, both direct and consensual, to light, better known as the Argyll-Robertson pupil. This is looked upon as one of the earliest as well as one of the most positive signs of developing

tabes, and is stated to exist in seventy per cent. of the cases. In forty-eight per cent. of the cases there are changes in the size of the pupils. Next in point of frequency, the external ocular muscles supplied by this nerve are involved, and among the symptoms of tabes ptosis takes first rank. It may present itself as an isolated single paralysis, or muscles innervated by other branches of this nerve may be involved; there may even be complete oculomotor paralysis, or ophthalmoplegia exterior and interior. According to some authors, ptosis associated with paralysis of the external rectus muscle is especially frequent in the early stages of tabes. Involvement of both external recti has been observed. Paralysis of the superior oblique has seldom been noted.

Case III.

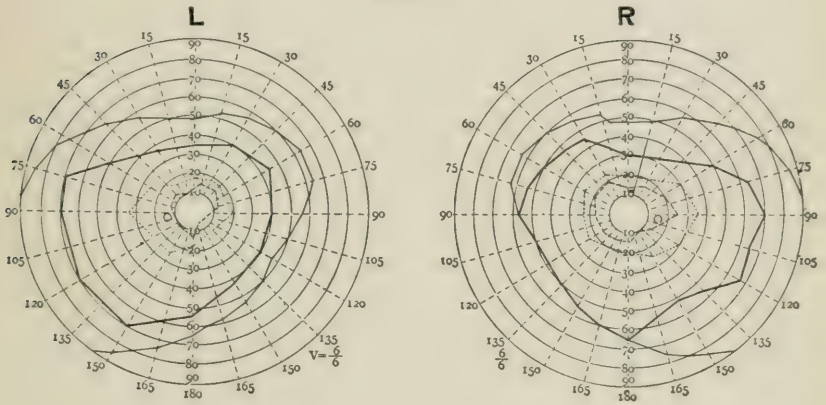


A sudden ptosis or paralysis of one of the extrinsic muscles of the eyeball in an individual over thirty years of age should at once arouse suspicion of some grave underlying disease. Modern pathology has taught us that any of the severer forms of infection attended by the development of toxins may lead to paralysis of the ocular muscles; but these causes may always be excluded by investigation of the history of the case. Many regard rheumatism, gout, and "taking cold" as causative factors, and such a pathogenesis is often confirmed by early disappearance of the paralysis. These symptoms are often fleeting. They appear, disappear and reappear. Often one nerve is restored in its integrity and some other branch is involved. Though it is true that each of these conditions may exist singly and be wholly independent of spinal disease, still, the

presence of one of them, especially if associated with but a single spinal symptom, such as loss or exaggeration of the patellar reflex, lancinating pains, or Romberg symptom, is sufficient to justify a diagnosis of tabes.

In the vast majority of cases the optic nerve is spared; the frequency of its involvement has been estimated at from seven to fifteen per cent. In most of these cases vision in the beginning is perfect, but a study of the visual field will disclose conditions which do not augur well for sight in the future. Cases with perfect fields for white disclose marked contraction for colors, especially for green and red; there may even be total loss of the color-sense. When a ten-millimetre square of red is called brown, or one of green is

Case IV.



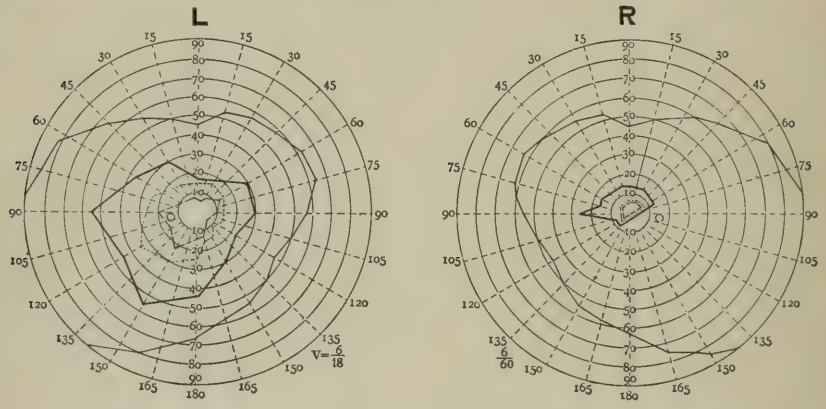
called yellow or gray, excluding congenital color-blindness, the diagnosis of retrobulbar disease of the nerve is warranted.

Color-blindness is a symptom of atrophy, and its rate of development is a measure of the progress of the disease. All the ganglionic cells or their axons in the retina are equally affected, and are no longer in a condition to transmit impulses even of great intensity.

It is assumed that the sensitiveness of the retina decreases towards the periphery. In the physiological condition it has been found that the field for blue is the largest; beyond it there is a narrow zone, in which, while the space-sense is still present, all objects appear gray. The field for red is narrower than the field for blue, and the field for green is narrower than that for red.

Schoen,[1] who investigated this subject in 1874, determined that the greatest nervous stimulus is required for the perception of green, a lesser intensity is required for red, still less for blue, until, under the weakest stimulation, gray is perceived. In other words, when the neurons of the retina are affected by disease they can no longer transmit an impulse of the intensity which is requisite for the recognition of the color green, while red and blue may still be perceived; if the degree of disease is such that an impulse of still less intensity can no longer be transmitted, neither green nor red will be perceived; finally, if the disease has progressed sufficiently, all objects appear gray.

CASE VII.



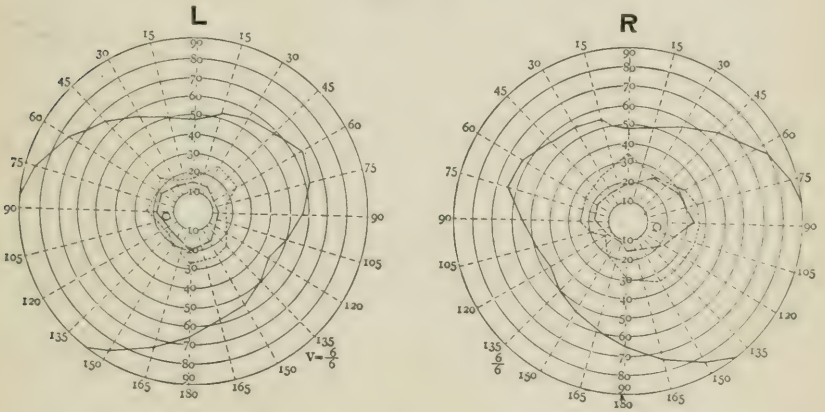
The study of the fields is invaluable, for not only the loss of the color-sense, but the contraction of the field for white also begins in the periphery, and the patient may be unaware for a long time that he is becoming blind, as the point of fixation is preserved to the last. When the disk has become pale, its outline well defined, and the lamina cribrosa clear, the diagnosis of simple gray atrophy is patent to any one who possesses sufficient skill to make an ophthalmoscopic examination. The diagnosis can be made long before this stage has been reached by a careful examination of the fields, especially for color. When marked contractions appear the prognosis is very grave indeed, but a diagnosis is but partially established until search is made for other signs confirmatory of tabes. (See diagrams of fields of Cases I., II., III., IV., VII., IX., XII., and XIII.)

[1] Die Lehre vom Gesichtsfelde, Berlin, 1874.

In some cases, for a long time only one eye is affected, but the other is sure to become involved later. At times the degeneration is rapid; again, it is very slow, finally leaving a very small central area in which vision is still preserved; but in all these cases the final result is hopeless blindness. It is a remarkable fact that though the ptosis and diplopia which are due to disease of the motor nerves may disappear, the changes in the optic, a sensory nerve, are permanent or progressive.

It has been recently asserted that this primary motor paralysis is merely a manifestation of loss of the muscle-sense, with a corresponding degree of incoördination of the ocular muscles. I have, however, met with cases of undoubted motor paralysis in which movement of the eyeball in the direction of the paralyzed muscle—

CASE IX.



the other eye being covered—was impossible. Inability to raise the eyelid can hardly be considered due to faulty judgment as to the amount of energy requisite to raise the lid to a certain height.

Reasoning from analogy, the existence of a sensory as well as of a motor paralysis of the ocular muscles may be inferred. And I would here incidentally call attention to the dangerous ground one is treading in making tenotomies in this class of cases.

This primary atrophy is never regressive, it is always permanent or progressive. There are those who assert that all cases of simple optic atrophy will eventually, if the patients live long enough, develop spinal symptoms. It is a strange fact, as yet unexplained, that those cases in which the ocular symptoms, especially

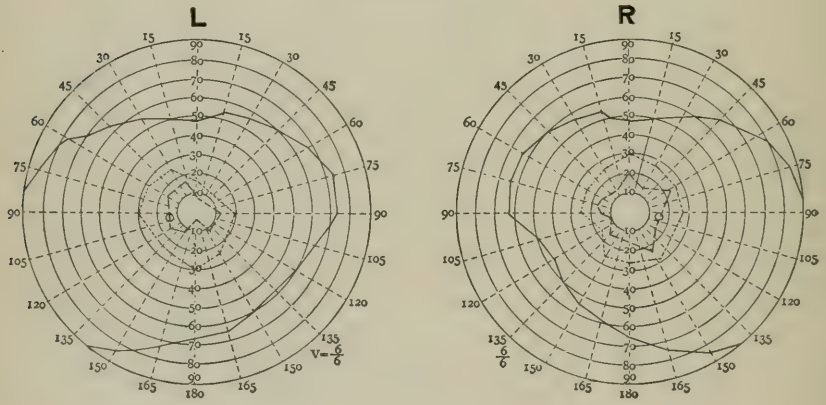
the optic atrophy, are especially severe, seldom develop the graver forms of spinal disease. A study of the fundus may add confirmatory evidence to a diagnosis still in doubt. Retinal and chorioidal changes may confirm the existence of syphilis.

The critical study of fourteen cases of spinal disease disclosed the following conditions:

Incipient locomotor ataxia in eight, tabes with cerebral involvement in four, and spinal lues in two.

Only two of these cases had exclusively ocular symptoms on which to base a diagnosis. All came under observation owing to ocular involvement. In thirteen, no previous diagnosis had been made, attention being first attracted to the underlying condition by the ocular symptoms. Twelve of these were males and two

CASE XII.



females, the ages ranging from thirty to sixty-seven years. In eleven cases syphilis was not denied, and the period since the date of infection varied from one to thirty-five years. But three cases had previously undergone antisyphilitic treatment, and these three cases disclosed most severe involvement of the nervous system. (See Cases II., IV., XI.) One case (V.) was due to exposure to cold and the carrying of heavy burdens; another (VI.) to a very severe blow on the neck; but the habits of life of both of these patients did not preclude the possibility of syphilis. In one case (XII.) no positive cause could be assigned.

The oculomotor nerve was involved in ten cases (I., II., IV., VI., VII., VIII., IX., XII., XIII., XIV.).

Ptosis was present in four cases (I., II., VI., XII.); and ptosis was combined with paralysis of the external rectus muscle twice (I., VI.).

The Argyll-Robertson pupil was present in four cases (II., IV., VIII., XII.).

Myosis in one case (I.).

Inequality of pupils, with but slight reaction, was present three times (I., VI., IX.).

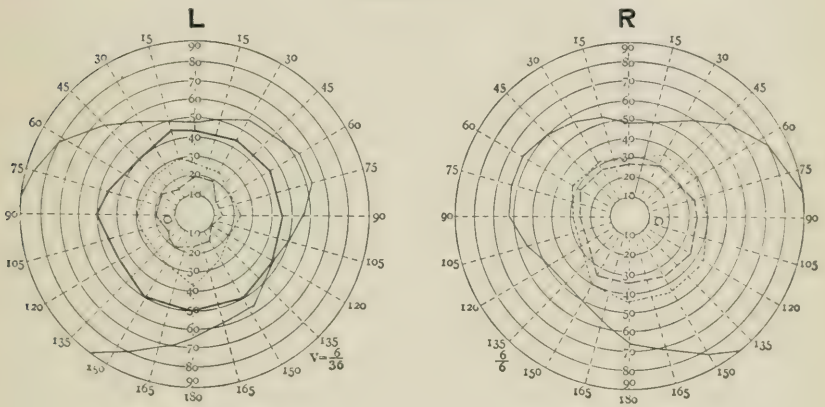
Reaction to light in but one eye once (I.).

Complete oculomotor paralysis once (II.).

Paralysis of sphincter of the iris twice (XIII., XIV.).

Paralysis of accommodation was present three times (II., XIII., XIV.).

Case XIII.



The fifth nerve was involved once (III.).

Paralysis of the external rectus was present in six cases (I., III., IV., V., VI., X.).

In one case (III.) both the external rectus muscles were paralyzed.

The seventh nerve was involved once (VII.).

Optic atrophy in its various stages existed in six cases (I., II., III., VII., VIII., XII.).

Hyperæmia of the disk was noted in three cases (I., II., XII.).

The color-sense was impaired in nine cases (I., II., III., IV., VII., VIII., IX., XII., XIII.), in eight of which there was contraction of the visual field.

Three of these cases, notwithstanding the contraction of the field, had normal vision (I., IX., XII.).

These cases, after a time, passed from my observation, and all that can be said of their course is that, notwithstanding antisyphilitic and tonic treatment, though some seemed for a time to improve, they subsequently relapsed and grew progressively worse. Not a single case was benefited by any mode of treatment.

Though tabes is looked upon more especially as a disease of the *sensory* nerves of the spinal system, this does not hold true of the cranial nerves, since in them one meets with initial lesions of *motor* as well as of sensory nerves; and also of nerves of special sense. In about fifteen per cent., possibly in a larger proportion of cases, the involvement of the cranial nerves is the first indication of a more extensive involvement of the spinal nerves.

One should never feel satisfied with the diagnosis of an isolated ocular paralysis until after a systematic examination for tabes or other forms of cerebro-spinal disease.

Simple gray atrophy of the optic nerve is essentially a degenerative process without previous inflammation,—a death of sensory neurons,—and should likewise arouse suspicion of tabes.

Finally, should not our treatment be directed to building up and strengthening the neurons rather than to attacking an underlying luetic or other toxæmia, from the results of which, rather than from its active presence, the entire organism may be suffering? I am fully aware that treatment should always be directed to the underlying cause, if one hopes to get results; but in tabes,—in fact, in all forms of nerve degeneration,—while one is treating the toxæmia the neurons die.

No. I.—I. S., March 3, 1893; age, thirty; male; causative disease, syphilis; *oculomotorius*—ptosis, R., moderate degree; iris, L., myosis, R., no reaction to light; sixth abducens, R., paralysis; fundus, R., hyperæmic disk; color-sense impaired, fields intact; vision, six-sixths, both eyes; patellar reflexes heightened; intolerable pruritus ani. *Incipient locomotor.*

No. II.—L. L., July 18, 1894; age, forty-two; male; causative disease, syphilis, fifteen years' standing. Was confined at Longview Asylum, February, 1893, to June, 1894. *Oculomotorius*—ptosis, L.; iris, Argyll-Robertson; pupil L. greater than R. Disk, R., hyperæmic, L., pale; color-blind for red and green; vision, L. = six-twelfths, R. = six-ninths. Loss of sexual desire; patellar reflex

absent. July 29, 1894, *oculomotorius*—ptosis lessened; iris, L. reacts, R. Argyll-Robertson continues; central scotoma for all colors; right field reduced to ten degrees. September, 1894, marked gastric crises; ataxic gait; great mental depression; relaxation of sphincters; patellar reflexes abolished. *Oculomotorius*—complete paralysis; vision, L. = six-eighteenths, R. = six-twelfths. 1895, symptoms were steadily progressive and mental condition weakening. November, 1895, vision, L. = six-thirty-sixths, R. = six-twelfths. 1896, tibia fractured by a very slight blow. April, 1897, declared insane; removed to Longview Asylum, where he died about one year later. *Locomotor and general paresis.*

No. III.—J. W., September, 1895; age, fifty-six; male; causative disease, syphilis, sunstroke in 1863; fifth trigeminus involved; sixth abducens, paralysis of both; fundus, disk ill-defined, gray atrophy; color-blind for red and green, contraction of both fields to less than ten degrees; vision, six-thirty-sixths, both eyes; ataxic gait, patellar reflexes gone, Romberg symptom present, grasp of hand weak, difficulty in controlling sphincter, lancinating pains in arms and legs, delayed sensation on face and in mouth from six to ten seconds, often has a burning sensation in mouth. *Locomotor and cerebral syphilis.*

No. IV.—F. B., February, 1896; age, forty-six; male; causative disease, syphilis twenty-one years ago; *oculomotorius*—iris, Argyll-Robertson; sixth abducens, R., paralysis; fundus, disk pale; normal form fields, contraction for colors; vision, six-sixths, both eyes; girdle sensation, lancinating pains. *Incipient locomotor.*

No. V.—H. K., March, 1896; age, sixty-seven; male; causative disease, exposure to cold and carrying heavy burdens; sixth abducens, L., paralysis; vision, six-eighteenths; lancinating pains in arms and legs, Romberg symptom. *Incipient locomotor.*

No. VI.—J. R., April, 1896; age, fifty-three; male; causative disease, blow on neck in 1891; *oculomotorius*—ptosis, left; iris, left pupil, $2 \times >$ right, some reaction to light; sixth abducens, L., paralysis; vision, six-sixths. *Incipient locomotor.*

No. VII.—L. A., May, 1896; age, forty; female; causative disease, syphilis; *oculomotorius*—iris, R., $2 \times >$ L.; seventh facial, paresis of left soft palate; fundus, atrophy of both disks; color-sense and field of vision, marked and irregular contractions; vision, L. = six-sixtieths, R. = six-eighteenths; pains in occipital region, gastric crises, lancinating pains, staggering gait, patellar reflexes gone. *Locomotor and cerebral basilar syphilis.*

No. VIII.—J. J. M., November, 1897; age, thirty-six; male; causative disease, syphilis for twelve years; *oculomotorius*—iris, Argyll-Robertson; fundus, atrophy of disk typical in both eyes; color-blind; vision, six-sixtieths, both; patellar reflexes gone, lightning pains, numbness of feet. *Incipient locomotor.*

No. IX.—L. B., June, 1899; age, forty-four; male; causative disease, syphilis for twenty-seven years; *oculomotorius*—iris, spinal myosis, slow consensual reaction; fundus, chorioidal patches in both

fundi; color-sense and field of vision, contractions for red and green; vision, six-sixths, both; heightened reflexes, loss of sexual desire for one and a half years. *Lues spinalis.*

No. X.—H. C., June, 1899; age, forty; male; causative disease, syphilis denied, but confirmed in offspring; sixth abducens, L., paralysis; vision, six-sixths; ulceration of nasal septum of many years' standing healed by potassium iodide, lightning pains, weakness in right arm, loss of patellar reflexes. *Incipient locomotor.*

No. XI.—C. G., July, 1899; age, forty-nine; male; causative disease, syphilis; fundus, cataract, incipient, in both eyes; vision, L. = six-eighteenths, R. = six-twenty-fourths; total paralysis of both lower extremities, extreme heightened knee reflexes, has had paralysis of both sphincters, no history of girdle sensation or lightning pains. *Lues spinalis.*

No. XII.—M. F., October, 1899; age, forty-six; male; *oculomotorius*—ptosis, fleeting; iris, Argyll-Robertson; fundus, L., disk pale, R., disk hyperæmic; color-sense and field of vision, marked contraction for colors; vision, six-sixths; patellar reflexes gone. *Incipient locomotor.*

No. XIII.—G. E., November, 1897; age, thirty; female; causative disease, syphilis and alcoholic excesses; *oculomotorius*—iris, L., paralysis of sphincter; accommodation, L., paralysis; color-sense and field of vision, L., contraction for red and green; vision, L. = six-thirty-sixths, R. = six-sixths. *Cerebral lues.*

No. XIV.—H. H., September, 1898; age, thirty-four; male; causative disease, syphilis for eleven years; *oculomotorius*—iris, R., paralysis of sphincter and accommodation; vision, L. = six-ninths, R. = six-twenty-fourths; hemiplegia two years ago, paralysis of left arm, girdle sensation, numbness in chest and arms, loss of patellar reflex. *Incipient locomotor.*

SOME CASES ILLUSTRATING DISEASE IN THE ETHMOID CELLS.

BY JOHN A. THOMPSON, M.D.,

Of Cincinnati, Ohio.

The anatomical relations of the ethmoid cells alone would justify the inference that disease in these cavities would profoundly affect adjacent organs. They are separated from the meninges and brain above by a thin plate of bone, one of the boundary walls of the cells. Anteriorly the floor of the frontal sinus is formed by the ethmoid cells, and the partition wall is very thin. The outlets of the posterior ethmoidal cells and those of the sphenoidal sinuses are in such close relation that the infection of the one from the other is easy. Externally the orbit and its contents are separated from the cells by a thin wall of bone in which there are occasionally dehiscences. The maxillary antrum is partially enclosed by this same external wall of the ethmoid cells. Internally the free surface of the ethmoid is covered by the mucous membrane of the nose, with which the lining membrane of the cells is continuous. The nasal mucosa, with its turbinated bodies, is, physiologically, more active than any organ of similar size in the body. In accordance with the pathological law that liability to inflammation is in direct proportion to physiological activity, the mucous membrane of the nose is more frequently inflamed than any other part of the body. It is reasonable to expect that this inflammation should often extend by continuity of tissue to the lining membrane of the ethmoid cells. (Figs. 1 and 2.)

We find nothing in earlier medical literature to indicate that this extension has been observed. Works that were authorities on diseases of the nose twenty years ago do not mention the ethmoid cells except briefly in the chapters on anatomy. Text-books on internal medicine that represent the knowledge of the profession a few years ago do not notice the ethmoid cells or their diseases, and we find no mention in them of suppuration in these cavities as a

possible cause of meningitis, abscess of the brain, or of general sepsis. Few if any of the latest authoritative books on the practice of medicine recognize the work done by rhinologists in this region. The pages of most text-books on surgery are equally blank when search is made for information concerning this topic. The best of the modern authorities in rhinology discuss the inflammatory diseases of the ethmoid cells, but they do not mention the obscure mental and nervous symptoms that are often produced by pathological changes in the lateral mass of the ethmoid, with its important relations. Modern rhinology teaches that the ethmoid cells are the most frequently diseased of the accessory sinuses. A thorough comprehension by the profession of the truths that observation has taught the rhinologist would make plain the diagnosis of many cases that are now obscure.

The common diseases of the ethmoid cells are inflammatory, infective, or neoplastic. The symptoms of acute catarrhal or suppurative inflammations are too well known to need repetition in an article designed to call attention to the rarer manifestations of disease in this region. The various forms of chronic suppuration are the cases in which nervous symptoms are most frequently observed.

Joseph S., a farmer, aged thirty-one years, had enjoyed perfect health until he had the grippe in December, 1898. From that time he suffered from constant headache. April 1, 1899, a new symptom appeared. He began to have an uncontrollable desire to sleep. This desire recurred daily at irregular hours, and would last from one to two hours. No matter what he was doing, when the period of somnolence came on he would immediately fall asleep. A thorough examination by his physician showed nothing abnormal except an hypertrophy of the right middle turbinated body and a slight exudation of pus beneath it. He came to me for operation September 26, 1899. The anterior two-thirds of the middle turbinated bone and one-third of the lateral mass of the ethmoid were found to be necrotic, and were removed by means of the snare, forceps, and curette. The symptoms were aggravated for one week after the operation. October 21, he reported that he was free from headache for the first time in ten months, and that he felt no abnormal sleepiness.

Chronic suppuration in the ethmoid cells may by infection pro-

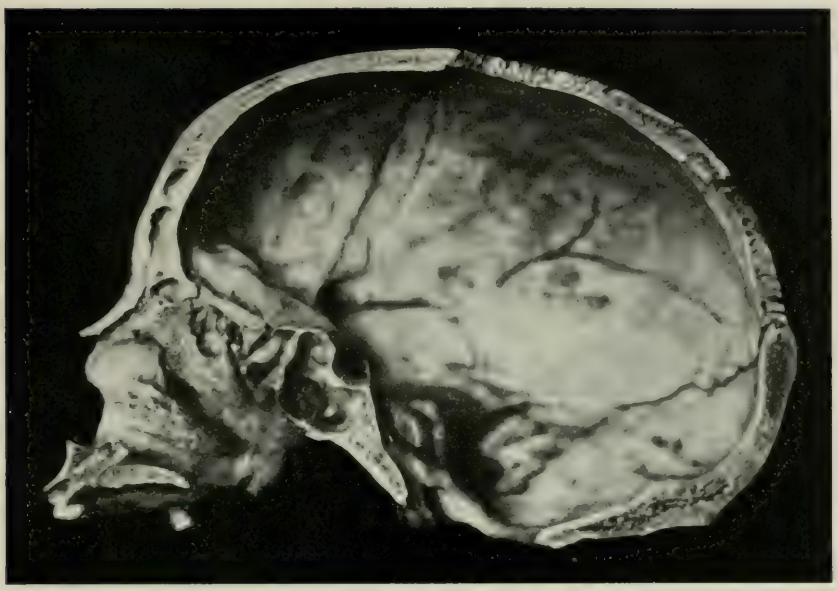


FIG. 1.—Antero-posterior section of skull, showing relations of ethmoid cells to the brain and to the sphenoidal sinus.

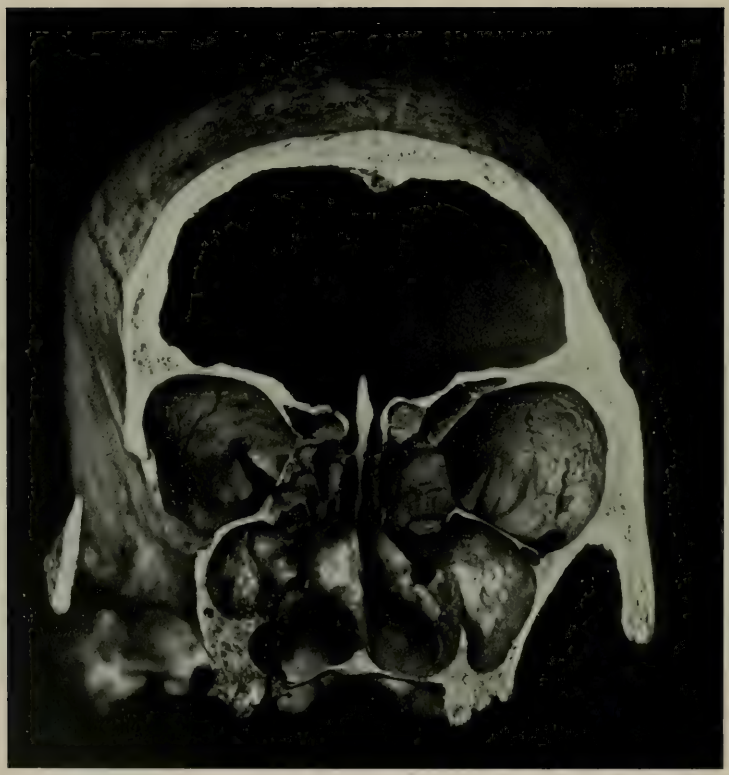


FIG. 2.—Transverse section of skull, showing relations of ethmoid cells to the frontal sinus, orbit, maxillary antrum, and nostrils.

duce inflammation of the meninges at the base of the brain. Joseph K., aged eighteen years, had suffered from atrophic rhinitis for seven years. Such treatment as he had received gave no relief. March 19, 1899, he began to vomit frequently and complained of severe headache. His temperature that evening was 102° F. These symptoms continued for three days, when the typical signs of acute meningitis developed. Headache at first was constant; later in the disease it became paroxysmal; the most acute pain was felt in the supra-orbital region. The temperature varied from 101° to 103° F. Vomiting was persistent throughout the attack. The patient would pass from delirium to stupor if an anodyne were given to relieve pain. He could usually be roused from either condition so as to answer questions intelligibly. The writer saw him first on the twenty-eighth day of his illness. His mind was then clear, and the muscles of the neck and back were no longer rigid. He had the irregular temperature of sepsis. The nose was found filled with offensive crusts of dried blood, mucus, and pus. Dead bone could be detected at the anterior end of the left middle turbinated body. Four days later this was removed under local anæsthesia, and the ethmoid cells opened so as to secure free drainage. There were an immediate fall of the temperature and a cessation of pain over the left eye. The fever returned the next day, but there was no pain over the left side. It was an error not to have drained the right ethmoid cells at the time the left were opened, but the patient's condition at that time was such that the least possible operative intervention seemed wisest. Local treatment was continued for the nasal disease, and general symptoms were met as they occurred. Convalescence was complete after nine weeks of fever. In September, when the patient's health was restored, the dead portion was removed from the right lateral mass of the ethmoid. This operation, with the home use of a spray, stopped the formation of offensive crusts in the nose. With free drainage of the cells the liability to a recurrence of meningitis is reduced to a minimum.

Chronic supra-orbital neuralgia is often caused by suppuration in the ethmoid cells. The amount of pus may be so small as to escape the notice both of the patient and the physician. An instance of this condition was presented by Mrs. S., aged fifty-five years, who consulted me in May, 1899. She complained only of a severe laryngitis. No mention was made of any nasal symptoms;

she had suffered from headache so long, and treatment had helped it so little, that she did not mention it, as she despaired of relief. I learned of this symptom only by direct questioning, after examining the nose for a possible cause of the laryngitis. The pain was often severe enough to confine her to bed for two days, the average duration of an acute attack. Some pain was always present. Examination of the nose showed a marked enlargement of the anterior portion of the left middle turbinated body. The septum was deflected and thickened, and pressed upon the right middle turbinated body. A very small amount of thick offensive pus was seen beneath the point of contact. An attempt to remove the growth in the left nostril revealed its nature. It proved to be a bony cyst due to distention of an ethmoid cell, which had extended into the middle turbinated bone. The cyst was three-fourths of an inch long and half an inch wide. Its removal gave partial relief to the headache. Removal of the thickened portion of the septum gave greater relief, but the cure was not complete until the right ethmoid cells were opened so that there was no obstruction to the escape of the pus.

A local lesion of tertiary syphilis seated in the ethmoid cells may by extension affect the meninges and cerebral cortex, and cause great mental aberration. In October, 1895, I was called to see C. H. W., aged thirty-four years. I had known him previously as a neat, alert, shrewd travelling salesman, a man of unusual ability. I found him in bed, unshaven, dirty, with dull eyes, cold, sallow skin, hanging cheeks, and open mouth,—the opposite in every particular to the man I expected to see. Two months previously he began to suffer from nasal stenosis on the left side. No attention was given this symptom. It was succeeded by a period of excitement, attributed by his friends to overwork and to his approaching marriage. This stage of excitement was followed by a period of depression, which soon developed into profound melancholia with suicidal tendencies. He refused for weeks to see any physician, but finally consented to let me examine his nose. The left nostril was completely closed by fungous granulations which bled at the slightest touch. A discharge of bloody pus into his throat caused an annoying cough. Necrotic portions of the left lateral mass of the ethmoid could be felt with the probe. The appearance was such that a diagnosis could not be made between syphilis and sar-

coma without a microscopic examination and the clinical test of antisyphilitic medication. The patient would answer questions intelligibly after considerable urging, but at this time denied a specific history. A portion of the obstructing mass in the left nostril was removed for microscopic examination and he was given potassium iodide internally. Cleansing sprays were ordered for the nose. The microscope was of little assistance in making a diagnosis, but the local disease in the nose improved under the internal treatment. The patient subsequently admitted specific infection four years before the present attack. The granulation tissue and dead bone were curetted out of the nose as soon as a diagnosis was made. The wound healed rapidly. Improvement in the mental condition was slower. He remained morose and apathetic. When urged by his employers to return to work he would not answer their letters. He remained in bed, but after prolonged solicitation would dress and come to my office. He made four attempts at suicide in as many weeks. He became impotent. A neurologist who saw him at my request made a diagnosis of cortical syphilis. When the ethmoid lesion had healed he was sent South. With change of environment and continued antisyphilitic medication he made a complete recovery. He resumed his work as a travelling salesman, and has had no recurrence of the melancholia. The presence of dead bone in the nose early in the case would indicate that the primary lesion had been in the ethmoid cells, with subsequent extension to the brain. The order of recovery would also strengthen the presumption that the nasal lesion was older than the cerebral. The case is not a parallel to the one of melancholia from ethmoid disease reported by Bosworth, but it is an additional example of the grave mental symptoms which sometimes follow the local lesion.

In every case of malignant tumor in the nose seen by the writer the point of origin has been the ethmoid cells. A recent case has suggested that a malignant tumor of the ethmoid may grow more rapidly into the maxillary antrum than into the nose. It will then produce all the symptoms of cancer of the upper jaw. If removed with the jaw by the usual method, enough of the tumor will be left in the remaining ethmoid cells to cause a prompt recurrence. It is possible that if the corresponding lateral mass of the ethmoid is removed with the jaw, the proportion of recurrences, stated by some authorities to be one hundred per cent., will be greatly les-

sened. The patient whose case led to this suggestion was Lena B., aged forty-two years. I saw her first in October, 1898. Her symptoms then were stenosis of the left nostril, with a bloody mucous discharge. At the time of menstruation the flow of blood from the nostril would be profuse, taking the place of the usual discharge. There was no local pain nor headache. The middle meatus was found filled with polypi. When an attempt was made to remove these with the snare, the bleeding was so free that the nose had to be packed before all the polypi were removed. At subsequent sittings, at intervals of two weeks, the remaining growths were removed, each operation being attended by free bleeding. While the middle turbinated region remained swollen, projecting into the nostril, the polypi did not return after removal. The oozing of blood from the nostril was not entirely checked by the operations on the polypi. It was found to come from a small growth under the mucous membrane in the floor of the inferior meatus. As this growth did not present the appearance of any common nasal neoplasm, no immediate operation was performed. It grew very slowly, but finally blocked the inferior meatus, and the hemorrhage from it slowly increased. It was curetted out and the wound healed promptly. In a month the growth returned. It was again removed, and a microscopic examination showed it to be an epithelioma. There was no recurrence in the nose, but early in June the patient came into the office with a perceptible fulness of the left cheek. It was then plain that while the growth of the tumor had apparently been controlled by intranasal surgery it was all the time extending into the maxillary antrum. The upper jaw was removed July 1. After it had been gotten out of the way the lateral mass of the ethmoid was cut out. The patient recovered promptly, and, with a rubber obturator, is able to teach school. It is much too soon to know the final result in this case. If we had stopped with the excision of the jaw, much of the tumor would have been left in the remaining portion of the ethmoid.

Patients with symptoms of constant supra-orbital pressure or pain, accompanied by mental dulness and depression of spirits, need a careful examination of the nose before the cause of their symptoms can be determined. Disease in the ethmoid cells with very slight local symptoms may aggravate or cause somnolence, despondency, or melancholia. Chronic anæmia may result from deep-seated sup-

puration in this region. The extent of malignant infiltration about the upper jaw, in the nose, or in the orbit cannot be learned without a thorough exploration of the ethmoid cells. Suppuration here must be excluded before the cause of any case of meningitis can be learned or the disease intelligently treated.

The cure of the obscure symptoms noted in these and other cases can be accomplished only by surgical intervention. The details of the operative treatment would be out of place in a paper intended only to call attention to a region whose pathology is usually ignored in diagnosis.

CHRONIC OTITIS MEDIA, RESULTING FROM CONTAMINATION IN THE TREATMENT OF ACUTE OTITIS MEDIA.

BY FRANK C. TODD, M.D.,

Professor of Clinical Ophthalmology and Otology in the University of Minnesota
Minneapolis.

Middle-ear inflammation followed by rupture of the tympanic membrane is of very common occurrence. It is due in a large proportion of cases to catarrh and other diseases affecting the nose and throat. It is particularly common in infancy and childhood. The inflammation is often of a catarrhal or of a mild septic character, the infection extending from the throat *via* the Eustachian tube. Many such cases recover without treatment or after simple syringing, while in others suppuration rapidly increases, the mastoid becomes involved, and death may result from extension of the inflammation to the brain, or the case may lapse into the chronic form, which always impairs hearing, and not infrequently causes total deafness. The suppuration is sometimes so profuse that the auditory canal has to be cleansed several times each day. The discharge is often foul, the patient is subject to earache, and the disease is considered such a menace to life that no life-insurance company will insure an applicant so affected.

In the treatment of middle-ear suppuration the necessity of removing growths and of treating malconditions in the nose and throat is well recognized; if this be attended to and aseptic treatment be carefully followed out, we may expect a speedy cure, for it is contamination that causes these cases to become chronic, and if there is no added or mixed infection the disease will run its course in a short time. An exposed tympanum, inflamed and secreting mucus or pus from the original infection, is a fertile field for the growth and development of germs, and the introduction of bacteria of a different variety may be followed by serious consequences. Says C. R. Holmes in regard to middle-ear inflamma-

tions:[1] "Whether the disease shall promptly run its course or change into the chronic form depends upon the continued activity of the germs present or upon the gradual dying out of the first culture and implantation of new varieties upon the now affected membrane."

The grave results of inflammation of the middle ear demand that the most strict precautions should be maintained against the introduction of sepsis or of bacteria of a new variety into an already suppurating ear.

The writer has for a long time regarded the piston syringe commonly used by aurists in the routine treatment of suppurations of the middle ear as a dangerous instrument. The cylinder is filled by suction, the nozzle being immersed in the medicated solution. When its contents are forced out the point of the nozzle touches the ear and comes in contact with the profuse discharge usually present. The syringe is removed, and used again on the next patient. In sucking up the solution this time some of the pus adhering to the point is drawn into the cylinder, and is forced into the ear of the next patient, or remaining on the point may cause direct infection. The leather washer, which cannot be boiled without being destroyed, also becomes contaminated and makes a good culture medium.

In the discussion upon this subject in the Otological Section of the American Medical Association, in June, 1899, one of the members stated that he did not allow the point of his syringe to come in contact with the ear. It is, however, almost impossible to prevent the point from occasionally touching, for patients cannot always be depended upon to keep quiet during the procedure. With the piston ear syringe contact must be most strenuously avoided, since we do not wish to communicate sepsis; but my observation leads me to believe that most aurists touch the point against the ear while syringing, for the direction of the current can be kept steadier by touching the entrance to the canal than by holding the point away. Even were the operator successful in holding the point away from the ear, it is possible for the return current to cause some of the septic material to be deposited upon the point of the syringe.

That germs are prevalent in quantity and in numerous varieties

[1] American Text-Book of Diseases of the Eye, Ear, Nose, and Throat, edited by de Schweinitz and Randall.

upon the point and upon the washer is evident from the bacteriological investigations made by the writer, the results of which are given below.

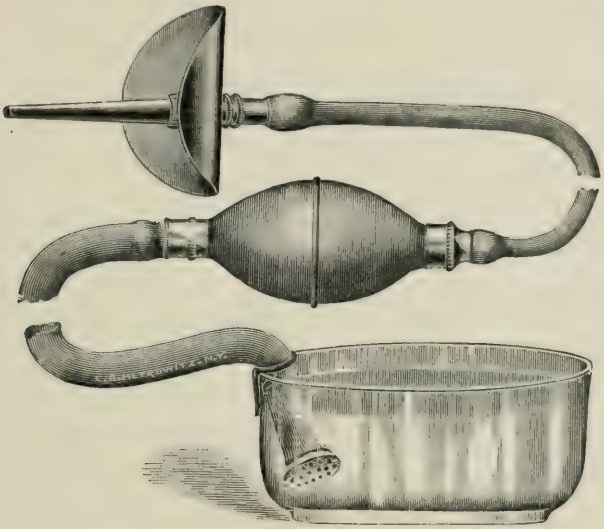
Many aurists favor the dry treatment of middle-ear suppuration, claiming better results; and doubtless in many cases better results are obtained than by using the ear syringe, because there is less danger of introducing septic matter in swabbing out with dry sterile cotton than in using the ordinary syringe which has to do duty for all suppurative cases. Harm is not likely to result from the introduction of sterile water, and much may be gained in washing away *débris* which cannot be removed with the swab or with instruments without much irritation. True, moisture is conducive to germ growth; but if moisture is already present the judicious employment of a non-irritating fluid like warm sterile water can do no harm, especially if the parts are thoroughly swabbed out with sterile cotton afterwards.

The germs that commonly cause inflammation of the middle ear are the diplococcus of pneumonia, the *Staphylococcus pyogenes albus,* the *Staphylococcus pyogenes aureus,* the *Streptococcus pyogenes,* and the *Bacillus pyocyaneus.* These and other germs found in the ear vary greatly in virulence. The staphylococci are, for instance, less virulent than the *Streptococcus pyogenes,* and the most destructive process results from a mixed infection by the *Bacillus tuberculosis* and the *Streptococcus pyogenes.* The ordinary less violent forms of chronic suppuration are due to the *Bacillus tuberculosis* and the *Streptococcus pyogenes.* The ordithe *Streptococcus pyogenes* is most frequently present.

In the investigations made by the writer, cultures were taken from the points and washers of five ear syringes belonging to five aurists, and the presence of various bacteria was demonstrated,—*e.g.,* the *Staphylococcus pyogenes aureus,* the *Staphylococcus pyogenes albus,* the *Streptococcus pyogenes,* the diplococcus of pneumonia, and the *Bacillus tuberculosis.* One of the syringes was sterilized and then used daily in routine treatment for two weeks. Cultures were then taken from the point and washer at intervals of two weeks while the following solutions were being used: sterile water, saturated solution of boric acid, bichloride of mercury (one to five thousand), carbolic acid (two and one-half and five per cent.). Cultures were obtained from both the point and

washer in every instance, though several attempts were sometimes necessary before a growth was obtained while the syringe was still in use. No doubt the solutions of carbolic acid and bichloride of mercury will in time destroy the bacteria, but they are of little or no value in preventing the conveyance of sepsis while the syringe is in daily use, as sufficient time does not elapse for the destruction of the bacteria.

An ear syringe, to be aseptic and practicable, must meet the following requirements: First. The point which comes in contact with the ear must admit of sterilization and be so constructed that it can be easily removed. Secondly. There must be no suction through this point.



The accompanying cut illustrates a syringe devised by the writer, which consists of a bulb, with a valve at each end, to which is attached a rubber tube, large at one end to permit the ready entrance of the fluid and thus insure rapid filling of the bulb, and small at the end through which the solution makes its exit. This smaller tube terminates in a point fitted with a shield, to protect the operator from the return flow. The point can be unscrewed and sterilized, and, having several points on hand, a clean one can be used for each case. If desired, the shield can be dispensed with, but it does not obstruct the view and the writer has found it a great advantage.

MASTOIDITIS AND OPERATIONS.

CLINICAL LECTURE DELIVERED AT THE CHICAGO POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL.

BY SETH SCOTT BISHOP, B.S., M.D., LL.D.

GENTLEMEN,—This afternoon you have seen six cases of mastoid inflammation resulting from various causes, and we will now turn our attention to mastoiditis as a complication and as a sequel of epidemic influenza, after a brief consideration of one patient of unusual interest. This girl, who is twelve years old, had scarlet fever in July, 1898. This was followed in two weeks by an acute suppurative inflammation of the middle ear and suppurative mastoiditis, rheumatism, nephritis, and endocarditis. On account of the heart and kidney complications, a general anæsthetic could not be administered for the purpose of opening the mastoid process, notwithstanding the evidence of the presence of an abscess; but one of my assistants made Wilde's incision down to the osseous fistula and liberated a considerable quantity of purulent discharge. The treatment of the heart and kidneys resulted so well that we were able to administer ether and perform the mastoid operation on the 7th of the following October. We found the mastoid process filled with pus, unhealthy granulations, and necrotic bone. All diseased or dead tissue was removed; the little girl rallied well, and the mastoid wound was kept open until it had completely filled with healthy cicatricial tissue, when it was allowed to close at the end of ten weeks. (Fig. 1.) I have seen this case only occasionally since the operation, but the dresser reports that she improved rapidly in general health from the time of the operation.

Another of the mastoid cases you have examined in this clinic has an extraordinary history. He is a private patient whom I have prevailed upon to give you the benefit of his experience. He is a travelling salesman, thirty-two years of age. In 1892, a neighboring aural surgeon performed a mastoid operation upon him for a suppurative mastoiditis complicating a suppuration of the middle

FIG. 1.—Mastoid wound three weeks after the operation.

FIG. 4.—Mastoid wound five weeks after the operation.

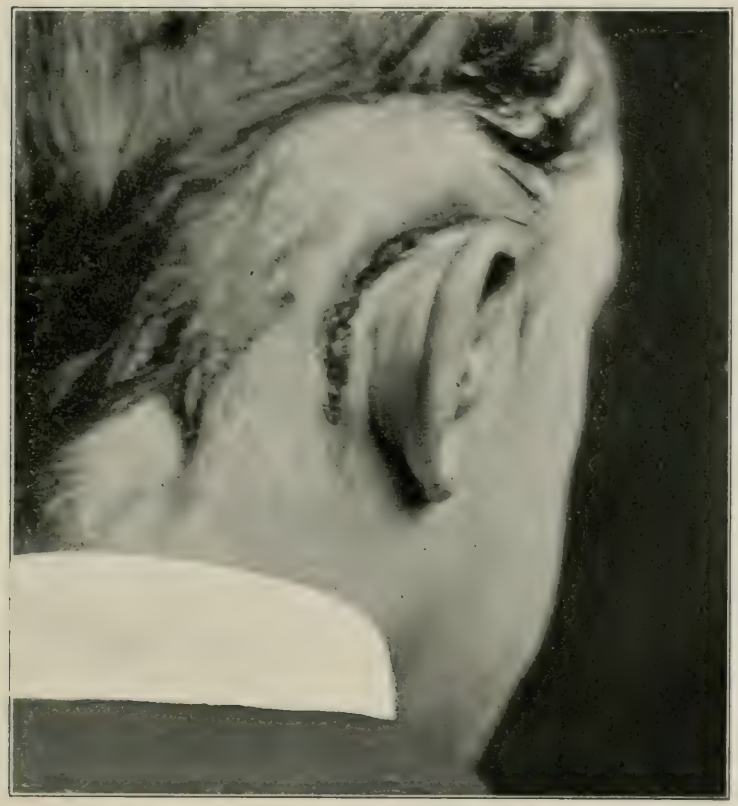
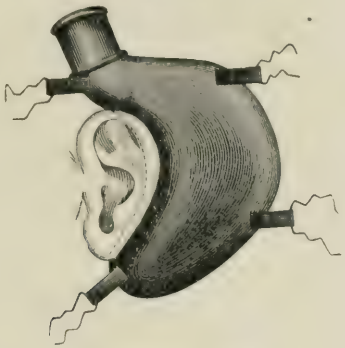


FIG. 3.—Mastoid wound two weeks after modified Stacke operation.

ear. The result of the operation was all that could be desired; the wound healed and the suppuration of the tympanic cavity ceased. He remained well until he became a sufferer from epidemic influenza, when the middle-ear suppuration returned and the mastoid process became inflamed again. Immediately we applied the mastoid ice-bag which I have devised for such conditions. (Fig. 2.) The pain, swelling, redness, and tenderness soon subsided.

Fig. 2.



Author's ice-bag.

On the same day that this man applied for treatment another case of acute mastoiditis resulting from influenza came. This was a university girl, eighteen years old, having a chronic suppurative inflammation of the middle ear of five years' duration. We will consider these two cases together on account of their striking contrast. The ice-bags were ordered to be applied in both cases the same day. After three or four days the inflammation had subsided in both mastoids. It happened that the young lady became greatly chilled one very cold night when the whole university was alarmed, and many of the students spent the night in heroic efforts to rescue several companions who had been carried out into Lake Michigan on an ice floe. This patient, supposing her brother to be one of the lost skaters, spent the night with other rescuers in the bitter cold. The mastoiditis returned, but the prompt and continual application of ice for two days to the inflamed process completely dissipated the inflammation. The ear and mastoid are now well, and the hearing is excellent.

The experience of the salesman was quite different. Without consulting me, he had his hair cut short during the inclement

weather, took cold, and had a sudden and violent return of the mastoiditis. It did not occur to him to apply the ice-bag, which had served him so well before. On his next visit there were great swelling, redness, tenderness, and pain over all of the mastoid region. We concluded to give the ice a trial, but it was too late to prevent the formation of pus. On making an incision through the tumor considerable pus was liberated, and the acute symptoms readily yielded to treatment. His ear and mastoid process are now well.

These two cases illustrate the utility of the ice treatment and the limits of its usefulness. When pus is not already present, disintegration of tissue and the formation of pus may be prevented. After ulceration of the mucous membrane lining the mastoid cells has occurred, or after pus has accumulated between the periosteum and the cortex, ice is inadequate to arrest the progress of the disease, and an operation is the only safe and curative procedure.

The fourth case is one of bilateral mastoiditis resulting from an attack of influenza. On the evening of January 19th last, I found Mrs. G., who is thirty-eight years old, suffering exquisite pain in both ears and mastoid processes. Both ears had begun paining her two days previously, and for thirty-six hours they had discharged copious amounts of "blood and water." Her temperature was 102.5° F.; the tongue was covered with a dirty-gray fur, was tremulous and indented by the teeth. There were also ominous symptoms of meningitis. The pupils were moderately dilated and sluggish, there were rigidity and tenderness of the muscles at the back of the neck, marked irascibility, mental aberration, and hyperæsthesia of the skin. Both drumheads were of a cherry-red color and perforated, œdematous, and bulging. The mastoid processes were red, swollen, and very tender. Mastoid ice-bags were applied to both processes, and the absolute continuousness of the effect of cold was insured by the alternating use of three bags. The third one was employed to prevent an interval of rise of temperature while each bag was removed to empty the accumulated water and to refill with ice, one bag being always filled and ready for application. It was necessary for the nurse to refill each bag every fifteen minutes, so great was the heat. In order to afford immediate relief from the violent pain, a combination of morphine, $\frac{1}{12}$ grain, atropine, $\frac{1}{100}$ grain, and caffeine, $\frac{1}{6}$ grain, was given sufficiently often to prevent suffering. The patient was kept in a partially recum-

bent posture in order to take advantage of the effect of gravity in reducing the volume of blood circulating in the head. The bowels were evacuated by a laxative, and a bland, unstimulating diet was prescribed. Local treatment consisted in gently drying the discharge from the auditory canals, inflation of the middle ears under as little pressure as sufficed to empty them, and the insufflation of aristol. A general amelioration of the symptoms promptly rewarded this treatment. The pain referable to the tympanic cavity and mastoid process soon ceased. The discharges diminished, the meningeal symptoms disappeared, and the patient appeared to be emerging from a siege of what threatened to be a battle for life with the odds against her. In a week the symptoms in one mastoid cleared up. The soreness was gone, and the ice was discontinued. A few days later the other process gave the same result. The ear treatment was continued, until now we are able to say that her ears and mastoid processes are well, and her hearing, she says, is as acute as it was before the attack. The ice-bags were required a longer time for this patient than in average cases.

The fifth mastoid case is now under treatment. It is another rare study, in that it presents a cholesteatoma of the tympanic cavity and mastoid process, and a neck abscess in a lad only fourteen years old, who has had a running ear for eight years. You saw me perform a modified Stacke operation on him and curette the mastoid and neck abscess, and I remarked that we would vary from the usual method of dressing the wound, with a packing of iodoform gauze, by allowing the cavity to fill with blood, and covering the seat of the operation as usual with iodoform gauze, sterilized cotton, and the bandage. You see the result two weeks after the operation. (Fig. 3.) The wound consists now of only a narrow curvilinear surface of healthy granulation tissue. There were a few exuberant granulations, which I repressed with the solid nitrate of silver. There is but a slight mucoid secretion from the ear, and no discharge whatever from the wound. This is a very pleasing and brilliant result. The boy was in a debilitated condition for a considerable time before the operation. He was just such a case as easily succumbs to an attack of epidemic influenza. (Since the operation he has regained normal health, his ear has been free from discharge, and his hearing has improved.)

The sixth case resembles the fourth in that symptoms of menin-

gitis developed in the course of the mastoiditis. It occurred in a boy of seventeen years, who had suffered from a chronic suppurative inflammation of the middle ear. The mastoid and meningeal symptoms were so urgent that I advised an immediate operation. It was too late to abort the formation of pus from an inflammatory process in the mastoid cells. On opening the mastoid process with the aid of the attending surgeon, Dr. B. B. Eads, I found it filled with pus, degenerative granulations of a dusky, fungoid appearance, and carious bone extending to the dura mater. All unhealthy and necrotic tissue was removed, exposing the dura and the wall of the sigmoid flexure of the lateral sinus. A brain abscess was suspected, but the use of the aspirating needle failed to disclose any pus-pocket. The wound was packed with gauze and dressed like the other cases, and a slow recovery took place. The boy's hygienic surroundings were bad, for he left the hospital and my service on the seventh day. Fig. 4 shows the appearance of the wound five weeks after the operation.

Incidentally, we will consider a seventh case, that of a merchant, fifty-four years of age, who is now under treatment. He has been subject for eight years to occasional attacks of middle-ear suppuration. During an attack of influenza he had an occasional purulent discharge from his right ear, for which irregular treatment was sought. Finally, he was prostrated, and called a physician. Besides his general illness, there was considerable pain in his ear, and a decided tenderness developed over the mastoid cells, but not over the antrum. On the next day, when he visited me, it appeared that the minute perforation in the anterior superior periphery of the drumhead did not afford sufficient exit for the greatly increased secretion in the middle ear. So I made a vertical incision through the posterior half of the membrane, extending from the upper to the lower periphery. This released a large amount of discharge which had evidently been stored up in the mastoid cells. Ice-bags were applied immediately and continuously, as in the fourth case, for two days and nights, when all mastoid symptoms vanished.

These cases amply illustrate the predilection of epidemic influenza for middle ear and mastoid complications of an intensely and rapidly destructive character, and the efficacy of ice when promptly and properly applied.

FIG. 1.—Impetigo contagiosa bullosa involving the whole body.

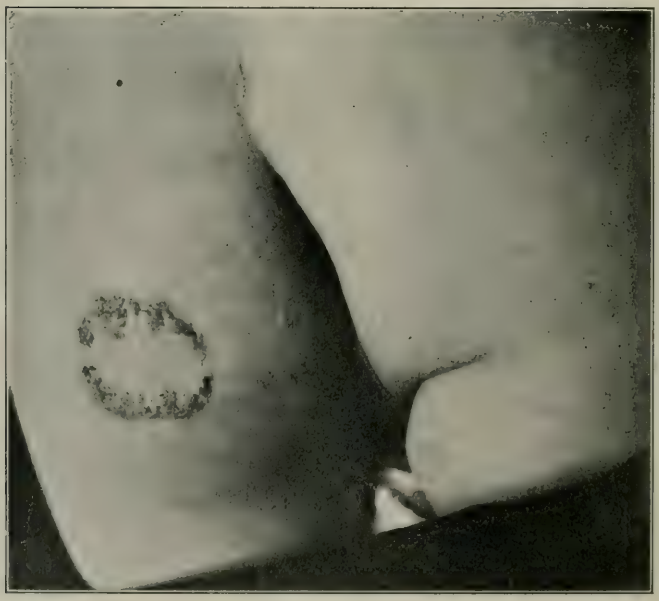


FIG. 2.—Impetigo of the thigh (posterior view). Ringworm form, or impetigo circinata: covered with crust at margin, centre free.

Dermatology

THE BULLOUS AND PUSTULAR ERUPTIONS OF EARLY LIFE.

CLINICAL LECTURE DELIVERED AT THE NEW YORK POST-GRADUATE MEDICAL SCHOOL, JUNE 1, 1900.

BY CHARLES WARRENNE ALLEN, M.D.,

Adjunct Professor of Skin Diseases in the New York Post-Graduate Medical School.

GENTLEMEN,—Desiring to speak to you to-day concerning the bullous and pustular eruptions of early life, I have selected from our rather large material such illustrative cases as have presented themselves during the past week. The first patient who claims our attention is this little girl of four years, who is affected with the bullous form of impetigo, or impetigo contagiosa bullosa. The lesions, as you note, are rather widely disseminated. There are rounded areas denuded of epidermis upon the face, marking the sites of bullæ whose walls have been brushed away; upon the hands there are large blebs arising, it would appear, from the healthy skin without even a reddened areola; while upon the feet and legs (Fig. 1) are scattered, as well as grouped, bullæ and pustules surrounding denuded areas, made up of small, discrete, rounded spots, caused by preceding bullous lesions. Here and there, we note an outline very suggestive of ringworm. In some cases the centre of the lesion clears up, leaving a ring-formed crust about the margin of the patch, which might make a careful differential examination necessary to exclude ringworm. In this connection I show you a photograph (Fig. 2) which well illustrates the similarity of the two affections. The diagnosis is made by finding the more typical lesions and crusts of impetigo elsewhere on the body, and by the absence of the trichophyton on microscopic examination.

If we were to study the lesions upon the feet and legs alone, without examining the rest of the body, we might find considerable difficulty in arriving at an exact diagnosis. The large plaques upon the dorsum of the feet, made up of separate bullæ which have ruptured, fused, and crusted, taken in conjunction with the rather large bullæ upon the legs, would suggest pemphigus, or possibly dermatitis herpetiformis. Now, there is an affection known as epidemic pemphigus, because of its characteristic tendency to spread, especially among the inmates of institutions, but also at times among children living in unhygienic quarters of a city, attacking nurses and mothers as well as those for whom they care. A study of these outbreaks has led me to believe that the condition is, in many instances at least, not pemphigus proper, but is in reality the same affection as that of which we now have an instance before us. An impetigo is, I believe, contagious, though some forms seem to be much more so than others. An epidemic outbreak depends simply upon the necessary conditions of propinquity or crowding and lack of care, especially in the exclusion of vermin. The *Pediculus capitis* is, to my mind, one of the most important agents in the dissemination of impetigo. This fact, as well as the great contagiousness of impetigo in certain instances, was recently exemplified in my private practice. A young man, of excellent family, presented himself with bullous and pustular impetigo of the face, neck, back, hands, and arms, together with marked pediculosis capitis, of which he was ignorant. A history was elicited of intimate association with a young lady who had subsequently been obliged to have her hair cut short because of pediculi and nits. A few days later another young man of equally good social position came to my office for the relief of virulent paronychial bullæ, implicating in succession almost all the fingers and following a "sore" on the chin. There were also lesions of impetigo upon the back of the hand, back of the neck, and upon the scalp, together with lice and their eggs in large numbers. His history was that he had spent several nights with his friend, Case I. Then came successively under my care the mother of Case I. and the father of Case II., the former showing pediculosis and beginning impetigo; the latter, a large solitary frank bulla upon the hand.

In the differentiation we are called upon to make between the so-called epidemic pemphigus of early infancy and syphilis bullosa,

Fig. 3.—Hereditary syphilis. Serpiginous lesions on the chin, patches and crusts upon the lips. "Mouth-breathing" from "snuffles," or occluded nasal passages.

FIG. 4.—Hereditary syphilis. Bullæ, macules, and crusts.

aside from the epidemic feature, we must take into account the usual freedom of the palms and soles in the former affection. Pemphigoid syphilis is usually of the congenital variety, and is of short duration; the lesions are dull, more purulent, and often of a greenish hue or hemorrhagic. The attendant never contracts the disease in this form, the bullæ appearing within a short time, as is not infrequently the case in "epidemic pemphigus."

Treatment.—The widely disseminated or universal form of recurrent bullous impetigo is a most obstinate affection. Unless we can succeed in hermetically sealing every lesion, after as thorough an application of pus- and germ-destroying agents as is possible, new bullæ will continue to appear. The wall of each bulla or pustule should be rubbed away with a three-per-cent. solution of methylene-blue by means of a firm, swab-like applicator, the solution being brought well to the limits of the undermined epidermis. This is a very different matter, so far as results go, from making applications over the unruptured wall of the bullæ or over the crusts *in situ.*

In order to kill germs the germicides must be brought in actual contact with them. A ten to fifty per cent. aqueous solution of ichthyol is an excellent application for limited areas. It should be allowed to dry, so as to form a varnish; it is then to be covered by layers of absorbent cotton held firmly in place by a bandage. The hands should be enveloped in a night-dressing, to prevent dissemination by scratching. For larger surfaces a ten to twenty-five per cent. watery solution may be applied by means of occlusive bandages. In every case of impetigo make sure that the head is freed from vermin and kept so.

I show you next an infant with a ring-like or serpiginous lesion upon the chin, resembling somewhat the crusted circles of impetigo (see Fig. 3). This, however, judging from the duller, less inflamed appearance and from the history and concomitant signs, is an instance of hereditary syphilis. There are sores and crusts upon the lips, and, because of the specific catarrhal condition known as "snuffles," there is pronounced and characteristic mouth-breathing. In contrast with the last-mentioned condition is this picture (Fig. 4 of a sleeping, specially infected infant, whose nasal passages were comparatively free, although the margins of the nostrils were implicated in the bullous and crusted lesions. Here, too, the color

of the intervening skin, as well as of the lesions themselves and of the crusts, was of a duskier, dirtier hue, the whole face presenting more of the *café au lait* tint so often mentioned by Diday and other of the older French writers. The resemblance which many of the vesico-pustular eruptions of infantile syphilis bear to impetigo, both in the location of the lesions about the natural orifices of the head and in the formation of rather thick crusts from the secretions poured out from their ulcerated base, makes the differentiation a matter of importance.

Pemphigoid lesions, common to both, as we have seen, complicate the matter still further, but we must bear in mind that in syphilis they affect the palms and soles, while in impetigo it is upon the back of the hand, especially the region about the nails, that the large bullæ are prone to occur. In syphilis there is also, though not invariably, a peculiar cachexia, and nasal catarrh is seldom absent.

Although I believe it unusual for late manifestations of hereditary syphilis to be the first to appear, I want to show you, in passing, this infant of two months, which has, so far as we can judge, escaped all outward manifestations of the disease up to the present time. You see it is a plump, fairly healthy looking child, though the mother is syphilitic. She presented herself with unmistakable evidence of the infection at about the fifth month of pregnancy, and we have kept her under almost continuous treatment ever since. She now comes for the relief of a painful, tender, bright-red, infiltrated, and rather hard swelling in the middle portion of the right thigh. This, beginning as a hard lump shortly after her confinement, has since grown painful, and recently became more inflamed. It presents the appearances of a localized phlebitis, and I have no doubt is of specific origin. We will at once resume the mixed treatment in full dose, and will apply over the swelling on the inner surface of the thigh mercurial ointment combined with ten per cent. of ichthyol. The infant will receive some slight medication through the mother's milk, and we shall watch for evidences of the disease. The history in this case is interesting, and I have mentioned it elsewhere. The mother acquired syphilis from her first child (this being the third), the child having acquired the disease from a nurse. After this accidental infection the mother became pregnant. Remaining under treatment, however, she was delivered of a living boy, who is now two and a half years of age.

At the age of two months he showed evidences of the disease, but was not treated for it. Nine months ago he came under my care for infiltrated moist patches, which began at the labial commissures and extended inward upon the buccal membrane. He was given vigorous local and general treatment, and the mother says he has remained well ever since. Now, to complete the story, the father of the child, the husband of our patient, came under treatment in February, 1898, for a chancre of the upper lip, whose origin he could not explain. Whether the wife or the son is to blame for the father's misfortune will never be known. The little patient before you is surely entitled to the disease, for now there is syphilis on both sides of the house. If later on he is found to be a victim, he can rightly claim that he inherits the affection from his older brother indirectly through one or both parents. The innocent originator of all this family trouble, himself a victim of innocent ignorance on the part of a care-taker, perhaps herself infected by some other child, died at the age of four years.

American Medical Association

THE 1900 MEETING OF THE AMERICAN MEDICAL ASSOCIATION.

BY GUY HINSDALE, M.D.,
Of Philadelphia.

The Fifty-first Annual Meeting of the American Medical Association was held at Atlantic City, New Jersey, June 5 to 8, 1900. It was the largest and most enjoyable meeting in the history of the Association. The famous resort never presented a more beautiful appearance. The welcome extended by the Committee of Arrangements and the attention to details providing for the comfort of the thirteen Sections made the meeting an unqualified success. Great credit is due to the President, Dr. W. W. Keen, of Philadelphia, to the Secretary, Dr. George H. Simmons, of Chicago, and to the Chairman of the Committee of Arrangements, Dr. Philip Marvel, of Atlantic City, for the admirable manner in which the scientific, business, and social programme was carried out.

The session was called to order by the President, Dr. Keen, and, after prayer by the Rev. Frederick J. Stanley and addresses of welcome by the acting Governor of the State and the Mayor of Atlantic City, Dr. Keen delivered his presidential address. The earnest, dignified manner of the speaker made a favorable impression upon all that were privileged to hear him.

In his address Dr. Keen dealt with a number of subjects of vital interest to the Association. He made a strong plea for the completion of the Rush Monument Fund, stating that at present there was in hand a little over eleven thousand dollars, a sum too large to go backward and not large enough to go forward.

The anti-vivisection bill introduced into the present Congress

was also considered, and a very strong letter from President Charles W. Eliot, of Harvard University, was read in connection with this topic. Dr. Keen spoke of the hearing given by the Senate Committee of the District of Columbia, at which arguments against the bill were made by Drs. William H. Welch, Henry P. Bowditch, H. A. Hare, William Osler, Mary Putnam Jacobi, George M. Kober, Howard A. Kelly, and B. E. Salmon, and by Surgeon-General George M. Sternberg, of the United States army, Bishop Lawrence, and the speaker. Dr. Keen thanked them, in the name of the Association and in the name of humanity, for their self-sacrificing help in opposing a measure which there is now reason to believe will either slumber in committee or be reported negatively. As President Eliot said, "It is the anti-vivisectionists who are inhuman and cruel to the last degree, because they would condemn both man and animals to suffering and death by impeding the progress of medical science."

Dr. Keen commended the establishment of a Section on Pathology and Pathological Exhibit, and invited the members present to inspect an exhibit which the secretary of the Pathological Committee, Dr. Frank B. Wynn, of Indianapolis, had collected from various parts of the country.

The President then took up the work of the different Sections, and advocated giving power to the editor of the *Journal* and to the trustees to discriminate in regard to the publication of papers presented at the annual meeting, the less important papers to be represented by abstracts.

Dr. Keen also recommended that financial support be given to the Committee on the Thirteenth International Medical Congress, to be held on August 2 in Paris.

He made a strong plea for the endowment of medical schools, stating that while each theological student in the United States enjoys the income from an endowment of two thousand two hundred and fifty dollars provided for his aid, each medical student has the income from only eighty-three dollars; and while he did not grudge a dollar to the theologians, he pleaded for his medical brethren, who, with a vastly more expensive education, should have also reasonable provision made for their training. He believed that the medical school which trained a Lister, a Pasteur, or a Koch has done more for humanity than all the hospitals in

this country combined. The modest laboratory at Würzburg consisted chiefly of a Ruhmkorff coil and a Crookes tube—and Röntgen. Other Röntgens and Listers we have among us, if we but knew it. These are the men who are the world's real heroes.

Dr. Keen advocated the establishment of grants of money by the Association to encourage scientific research in the United States.

The Executive Committee of the Congress reported favorably upon the recommendations of the President, and this Association will immediately be benefited thereby.

Of the papers presented at the general meeting of the Association the one which attracted the greatest attention after the President's address was that delivered by Victor C. Vaughan, Professor of Hygiene in the University of Michigan, and late major and division surgeon of United States volunteers. A committee consisting of Professor Vaughan, Major Walter Reed, of the United States army, and the late Major E. O. Shakespeare, of the United States volunteers, was appointed, at the request of the Surgeon-General of the United States army, to ascertain the causes of the existence and spread of typhoid fever in the national encampments and to suggest means for its abatement.

Major Vaughan made several remarkable statements, to which we particularly call attention:

Every regiment (both regular and volunteer) in the United States service in 1898 developed typhoid fever. More than ninety per cent. of the volunteer regiments developed the disease within eight weeks after assembling in the State encampments. Most—probably all—of the regular regiments developed typhoid fever within less than eight weeks after going into camp. It also became epidemic both in encampments of only one regiment and in those comprising more than one corps. Typhoid fever became epidemic in camps located in the Northern as well as those located in the Southern States, showing that the epidemics were not due to geographical location. Typhoid fever usually appears in military expeditions within eight weeks after assembling, as shown by the experience of the armies in South Africa in 1877 and 1878, in the Zulu war of 1878, in the Afghan campaigns of 1878 and 1880, in the Egyptian expedition of 1882, and in the Nile campaigns of 1884 and 1885.

Neither the miasmatic theory of the origin of typhoid fever nor the typhogenic theory is supported by the committee's investigations. Professor Vaughan believes that one or more men already infected with typhoid fever enlisted in nearly every command. The theory that the colon bacillus may be transformed into the typhoid fever germ is disproved. The history of typhoid fever in every encampment shows not only that this disease was not evolved from simple diarrhœa, but that, as a rule, the men who had diarrhœa did not subsequently develop typhoid fever. The investigations confirmed the doctrine of the specific origin of typhoid fever. With the disease so widely disseminated in this country, the probabilities are that if a regiment should be assembled in any section, and kept in a camp the sanitary conditions of which were perfect, one or more cases of the fever would develop. Typhoid fever is disseminated by the transference of the excretions of an infected individual to the alimentary canals of others. The disease is more likely to become epidemic in camps than in civil life, because of lack of facilities for disposing of fecal matter. A man suffering from typhoid fever may infect every latrine of a regiment before the disease is recognized in himself. Infected water was not an important factor in the spread of typhoid fever in the national encampments in 1898. Flies undoubtedly served as carriers of the infection. Personal contact was undoubtedly one of the means by which the infection was spread. It is probable that the disease was disseminated to some extent in the form of dust. A command infected with typhoid fever is not freed from the infection by simply changing location. When a command in which typhoid fever is prevalent changes its location, it carries with it the specific agents of the disease in the bodies of the men, and in their clothing, bedding, and tentage. After a command becomes badly infected with typhoid, change of location, together with thorough disinfection of clothing, bedding, etc., is necessary. An ocean voyage does not relieve an infected command of the disease. Except in cases of most urgent military necessity, one command should not be located on a site recently vacated by another. In military practice typhoid fever is often apparently an intermittent disease. The belief that errors in diet with consequent gastric and intestinal catarrh induce typhoid fever is not supported by the

investigations. The belief that simple gastro-intestinal disturbances predispose to the disease is not confirmed by the investigations. More than eighty per cent. of the men who developed typhoid fever had no preceding intestinal disorder. The deaths from typhoid fever were more than eighty per cent. of the total deaths. The shortest period of incubation in typhoid fever is probably under eight days. One who has lived in a camp in which typhoid fever is prevalent is liable to develop this disease at any time within eight weeks after leaving such camp.

Dr. George Dock, of Ann Arbor, in his address as President of the Section on the Practice of Medicine, stated that it is a discouraging fact that, although medicine has made most remarkable advances in this century, its influence over the layman is decreasing rapidly. While it is becoming more positive, more candid, and more accurate, the grossest delusions flourish. Not only the plausible nostrum-maker, but the palmist and the astrologer have their believers by the thousand. Dr. Dock commented on the weaknesses of the profession, the commercialism of the day, and the prevalence of therapeutic false prophets.

Dr. William L. Rodman, of Philadelphia, delivered the address on surgery before the Association, choosing for his subject "Gastric Hemorrhage." This subject interests surgeons and medical practitioners alike. The speaker asserted that while ice, astringents, and opium, with rest, might arrest the first and second hemorrhages, surgical measures should be resorted to before a fatal attack ensued, and he demonstrated the analogy between this disease and appendicitis. The preferable time for operation is between the attacks, and this should be done if the first two attacks are close together. Dr. Rodman advised the use of hot water by enemata and in small quantities by the stomach. He stated his belief that it will be both less likely to excite vomiting and more certain in its hæmostatic effects than ice. In the present state of our knowledge we cannot say that operation should ever be done during the first hemorrhage or the ensuing shock. Likewise, if seen after hemorrhage, when the patient is rallying from shock, the policy of non-intervention is not only permissible but best. If the hemorrhage is rapid within a short time, the question is, Shall we operate? Dr. Rodman stated that there have been thirty-two operations for acute hemorrhage and thirteen deaths.

He presented a table, prepared by Drs. W. Hersey Thomas and Stilwell C. Burns, giving the results in sixty-three cases of acute and chronic gastric hemorrhage that had been operated upon. Dr. Rodman gave the result of experimental operations upon four dogs, by which he endeavored, if possible, to cause hemorrhage into the stomach by rapid and severe traumatism, not applied to the stomach itself. He then took up the subject of hæmatemesis after various surgical operations. All septic conditions favor disintegration of the blood-vessels and free hemorrhage from mucous surfaces. The gastric mucosa is particularly liable to congestion in conditions of sepsis, both on account of the marked tendency of the thin and more or less disintegrated blood to settle in the internal organs and the vomiting and retching so frequently present.

Dr. George M. Gould, of Philadelphia, read a paper on "A System of Personal Biological Examinations the Condition of Adequate Medical and Scientific Conduct of Life," in which he stated that too few biological examinations of human beings are made. Sometimes examinations are made by universities in the case of athletes, but it is not common to keep correct data for future reference. If positive psychological and physiological measurements were made at regular intervals, disease in its earliest stages might often be detected. Specialists are encroaching too much upon the field of the general practitioner, so that in time the latter may have the narrowest specialty of all.

Dr. C. N. B. Camac, of New York, read a paper on "The Hospital Clinical Laboratory." The workshop of the practitioner is a combination of the sick-room, the bedside, and the ward. Ward laboratories should be established and fitted up with an oil immersion lens, an alcohol lamp, facilities for running water, etc. Wherever ward laboratories had been established they had proved of the greatest advantage. Fifty dollars a year has been found ample for their maintenance.

Dr. M. Howard Fussell, of Philadelphia, read a paper on "The Examination of the Blood: its Value to the General Practitioner." In certain cases he believed that more can be learned from a blood examination than from testing the urine. As a general practitioner he examines the blood in all obscure cases of disease.

Dr. Simon Flexner, of Philadelphia, read a paper on "Dysentery." Blood-serum taken from a case of the acute form of the disease agglutinated the pure culture of the bacillus of Shiga. This bacillus seems to be more frequently found in the acute cases, while the amœba is present in the chronic forms of dysentery of the tropics.

Dr. John H. Musser read some "Notes on Tropical Dysentery." He reported the case of a soldier who died in Philadelphia after returning from Porto Rico. In this case the agglutinative test with the bacillus of dysentery, isolated in the Philippines and illustrated in the April CLINICS, was positive. In the discussion of these papers Dr. William Osler, of Baltimore, gave it as his opinion that the bacillus of Shiga was a more important factor in the production of dysentery than the amœba coli.

Dr. J. C. Wilson, of Philadelphia, read a paper on "Serumtherapy in Croupous Pneumonia." He gave the results obtained in a series of cases treated with and without the serum. Large doses had to be employed to get any results. While it might have a favorable outlook, the results obtained showed that serumtherapy does not shorten the duration of the disease, nor cause anticipation of the crisis. Twenty cases were treated, with four deaths.

Dr. T. J. Happel, of Trenton, Tennessee, read a paper on "Pseudo- (?) or Modified (?) Small-pox." Two hundred cases had been studied. The usual early symptoms of small-pox had been present, such as backache, headache, and fever; but as soon as the eruption appeared the patient seemed greatly relieved. The pustular stage of true small-pox was absent. In some of the first cases seen the patients had not been vaccinated. Dr. Walsh, of New York, stated that Senator had advanced the opinion that there was a disease intermediate between varicella and variola. Varicella, in the opinion of Senator, was variola modified through the passage of many generations. Dr. McCormack believed that doubts as to a diagnosis might lead to a neglect of proper precautions against small-pox, and hence give rise to an epidemic of this disease. He believed that the cases reported were those of small-pox.

Dr. Eugene Wasdin, surgeon in the United States Marine Hospital service, read a paper on "Yellow Fever: its Nature and Causes." According to his observations and experiments made in

the laboratory, the *Bacillus icteroides* is the cause of yellow fever. He had been able to isolate it in 85.7 per cent. of the autopsy cases. The bacillus X of Sternberg was a distinct micro-organism from the *Bacillus icteroides*. The *Bacillus icteroides* is an artificially pathogenic organism. Below the frost mark it has a tendency to lose its pathogenic properties. He believed that the organism gains entrance into the system through the respiratory tract. Dr. Simon Flexner thought that the question as to the *Bacillus icteroides* being the cause of yellow fever must be further confirmed by different observers.

The afternoon of the second day was devoted to a symposium on malarial fever. Dr. W. S. Thayer, of Baltimore, read a paper, entitled "Etiology of Malarial Fever with Especial Reference to Mosquitoes." The speaker detailed a series of personal investigations in regard to determining this question. He first spoke of the cycle of development of the malarial parasite within the human body and in the tissues of the mosquito—*Anopheles*. While there may be other modes of introduction of the parasite into the body than by inoculation, the latter method is the only one which has been proved. The early spring cases may be regarded as relapses, while those occurring during July result from the bite of the *Anopheles*. To prevent the spread of malarial fever the patient should be protected with a mosquito-bar.

Dr. L. O. Howard, of the United States Department of Agriculture, gave an interesting lantern demonstration, showing the differences in the appearance and the development of the *Culex* and *Anopheles*. The larva of *Anopheles* lies flat upon the surface of the water and feeds in this position, while the larva of *Culex* floats with head downward and breathes through a pneumatic tube at the end of its tail. By this means the two genera may be distinguished. The "hum" of the *Anopheles* is about four tones lower than the *Culex*. When the *Anopheles* rests against the wall the body projects outward, while in *Culex* the body hangs downward. The *Anopheles* has spotted wings, while the wings of the *Culex* are uniform in color.

Dr. Albert Woldert, of Philadelphia, read a paper, entitled "Inoculation of Malarial Fever through the Agency of the Mosquito," and exhibited some sections and dissections of the mosquito. The speaker reviewed the history of the subject and also

the manner of development of the zygotes of the parasite in the middle intestine and venomo-salivary glands of the mosquito. Charts were exhibited showing that certain differences existed in the anatomy of the *Culex* and *Anopheles*. For the destruction of mosquito larvæ in their later stages (pupæ stage) a strong solution of tobacco in kerosene oil had proved most efficient.

A paper by Dr. Charles F. Craig, of the United States army, entitled "Some Typical Cases of Estivo-autumnal and Tertian Malarial Fevers, with a Study of the Parasites observed in the Blood," was read by Dr. Futcher. The writer concurred in the views of certain Italian authors that there are two types (quotidian and tertian) of the estivo-autumnal parasite. He also exhibited the temperature charts showing the differences found in infection from the quotidian and tertian estivo-autumnal variety of plasmodia.

Dr. Frank Jones, of Memphis, read a paper on "Clinical Observations in Malaria," in which he insisted on the value of clinical as opposed to microscopic diagnosis. The negro seems to be slightly immune to malarial fever. Brunettes are found to be more susceptible to malarial infection than blondes. Obesity is common in people residing in a malarial district.

Dr. William Britt Burns, of Deckerville, Arkansas, read a paper on "Malarial Hæmoglobinuria based on Sixteen Cases in which a Microscopic Examination of the Blood had been made." He regards quinine as the proper treatment in such cases.

In discussing these papers Dr. Krauss, of Memphis, Tennessee, said that hæmoglobinuria is a syndrome, and that it is not a mere symptom. In his opinion, the condition is not a toxic one. Some substance must be in the circulating blood in order to cause the condition, since quinine will at times produce it, while at other times it will fail to do so.

Dr. Robert B. Preble, of Chicago, read a paper on "Prolonged Fevers of Obscure Origin." Since the introduction of the Widal and diazo reactions, more careful examinations of the blood for malarial parasites, the exclusion of septic conditions, malignant heart disease, etc., the field of obscure fevers has become lessened.

The morning of the third day was devoted to a symposium on the subject of Arthritis.

Dr. David Riesman, of Philadelphia, read a paper on the "Pa-

thology of Rheumatism," noting the following facts as indicating its infectious origin: (1) fever; (2) chill; (3) epidemic outbreaks; (4) seasonal and climatic influence; (5) character of the complication; (6) a demonstrable portal of entry for the virus in many cases, as the tonsils, otitis media, operations on the nose, furuncles, vaccination, etc.; (7) occurrence of endocarditis; and (8) the direct transmission from mother to fœtus.

Dr. James J. Walsh, of New York, read a paper on "Rheumatism and the Prevention of Heart Complications." In his opinion, rheumatism is not a distinct entity but a series of allied processes. The acid reaction of the blood might be of a conservative nature. The use of salicylates does not lessen the tendency towards heart complications.

Dr. Charles W. Burr, of Philadelphia, spoke on "The Relations of Chorea and Rheumatism." He stated that frequently there was a history of rheumatism in cases of chorea. Chorea seldom occurs during an attack of rheumatism. The fact that the two diseases may coexist would not justify one in saying that one had more than a predisposing influence over the other.

Dr. De Lancey Rochester, of Buffalo, read a paper on "The Heart in Rheumatism." He believes that the process is undoubtedly infectious.

Dr. A. O. J. Kelly, of Philadelphia, spoke on "The Pathogenesis and Clinical Features of Arthritis Deformans." In his opinion, the disease is a tropho-neurosis the bacteria of which may have a selective tendency to affect certain joints, thus accounting for local lesions. The acute cases seem to be of an infectious type.

Dr. F. A. Packard, of Philadelphia, in discussing these papers, stated that rheumatism is not a local disease, but a series of symptoms produced by an unknown factor, with a tendency to involve the serous membranes of the joints and the endocardium. Arthritic diseases may be classified under six headings, as follows: (1) acute articular rheumatism; (2) acute infectious arthritis, as in gonorrhœa and scarlet fever; (3) chronic infectious arthritis, as in children; (4) rheumatoid arthritis; (5) spinal arthropathies; and (6) arthritis occurring in gout. The disease is an infectious one, and the tonsils are a favorite site for the entrance of micro-organisms. Dr. J. H. Musser, of Philadelphia, believed the disease to be an infectious one. Dr. J. M. Anders,

of Philadelphia, thought the disease undoubtedly infectious. The endocarditis of chorea is not necessarily rheumatic endocarditis. Dr. L. F. Bishop believed that in rheumatism there is a disturbed metabolism the nature of which is as yet unknown. In the joint cases infection was doubtless the cause. In the treatment, iron and strontium salicylate were advocated. "A Case of Malignant Endocarditis with Recovery" was reported by Dr. N. S. Davis, Jr., of Chicago.

Dr. Solomon Solis Cohen, of Philadelphia, read a paper on "The Relative Importance of Valvular and Muscular Lesions in Diseases of the Heart." In his opinion, the state of the cardiac muscle was of more importance from a prognostic point of view than the location of the lesion. In the early stages the symptoms of cardiac disease are not marked. He thought that influenza was a very common cause of heart disease. For this affection he advocated massage, regulation of the diet, and carbonated baths.

Dr. Louis Faugères Bishop, of New York, spoke on "A Clinical Study of Myocarditis." He believed that the myocardium was of as much importance as the pericardium or endocardium. Two clinical groups may be recognized: (1) myocarditis due to infectious processes, and (2) myocarditis due to diseased blood-vessels. In the treatment complete rest was advocated.

Dr. J. J. Morrissey read a paper on "A Plea for a more Rational Prognosis in Cardiac Affections." In order to render a truthful prognosis, it is essential that a careful diagnosis be first made. In ascertaining a diagnosis one should also consider the cardiac efficiency, whether or not there is hypertrophy, and whether or not the distant organs are functionally disturbed. Murmurs in themselves do not always forebode an unfavorable prognosis. In young athletes cardiac hypertrophy may be outgrown. Persons with aortic stenosis sometimes live to old age. Dr. James B. Herrick, of Chicago, stated that the presence of a heart-murmur during the course of rheumatism might be due to myocarditis, endocarditis, or changes in the blood. Dr. M. Howard Fussell spoke of instances in which a murmur might be transmitted to the vessels of the neck without the presence of aortic stenosis.

Dr. W. Freudenthal, of New York, read a paper, entitled "In what Relation does Occupation stand to Tuberculosis?"

Dr. J. M. Anders, of Philadelphia, read a paper on "Diag-

nosis and Treatment of the Prebacillary Stage of Pulmonary Tuberculosis." This condition is learned by the skiagraph, hypodermics of tuberculin, and lastly a very careful and accurate history. In most cases an evening exacerbation of temperature will be found.

Dr. Carroll E. Edson, of Denver, described "The Importance of Rest in Pulmonary Tuberculosis."

Dr. C. P. Ambler, of Asheville, North Carolina, read a paper on "Pulmonary Tuberculosis: Present Condition of Cases treated during 1898 and reported Last Year at the Columbus Meeting." A large percentage of these appeared to be permanently cured.

Dr. A. F. Lemke, of Chicago, in his paper on "Tuberculosis of the Lungs," advocated the intra-pleural injections of nitrogen.

Dr. Thomas J. Mays, of Philadelphia, read a paper on "The Treatment of Pulmonary Tuberculosis by the Injection of Silver Nitrate."

Dr. James B. Herrick, of Chicago, spoke on "The Diagnosis of Diabetes." Besides the presence of glucose, impotence and psychic disturbances may often be noted.

Dr. Heinrich Stern, of New York, read a paper, entitled "Mortality from Diabetes Mellitus in New York during the Decade 1889-1899." In 1860 fifty per cent. of the deaths were females. The mortality seemed worse in the fall of the year than at any other period. Diabetes is rare in childhood and adolescence. The mortality in negroes is low. Dr. M. B. Hartzell, of Philadelphia, described the cutaneous diseases accompanying diabetes. The importance of the recognition of cutaneous disturbances as an indication of general diseases should be learned by the general practitioner. In diabetes the skin would usually be found to be dry, the hair lustreless, and the nails brittle. Eczema and pruritus were frequently encountered.

Dr. O. T. Osborne, of New Haven, spoke on "Exophthalmic Goitre." In the treatment he advised strophanthus with mental and physical rest. Ordinarily an operation was not justifiable. Dr. Solomon Solis Cohen advised one-two-hundredth of a grain of hyoscine hydrobromate and one-twentieth of a grain of picrotoxin every four hours. Thymus feeding might also be tried.

Dr. James M. Peck, of Arlington, Kentucky, read a paper on "The Treatment of Typhoid Fever with Bacterial Products in

Connection with Other Agents." In the discussion of the above paper Dr. Wasdin expressed the belief that the typhoid germ gained entrance most frequently through the respiratory tract.

SECTION ON SURGERY AND ANATOMY.

The annual address was delivered by the chairman, H. O. Walker, of Detroit. The speaker detailed the advances made in surgical operations for both benign and malignant troubles. He had used Senn's plates and the Murphy button with equally good results. He advised an exploratory incision in all obscure cases.

Dr. W. L. Rodman, of Philadelphia, read a paper on "Non-perforating Gastric Ulcer with and without Hemorrhage." In Germany gastric ulcer is said to occur in five per cent. of the cases coming to autopsy. From results obtained by Welch in seven hundred and ninety-three cases, the lesser curvature, posterior wall, and pylorus were involved three times more frequently than other portions. Multiple ulcers have also been found. The most characteristic symptom was a severe and boring pain localized and influenced by food. The treatment at first should be medical. The operations indicated were pylorectomy and gastro-enterostomy. The Murphy button might be used in the latter operation.

"The Diagnosis and Treatment of Cholelithiasis" was the title of a paper read by Dr. W. J. Means, of Columbus, Ohio. He reported twenty cases, in which ten had been operated upon with no deaths. In three of those not operated upon death ensued.

W. J. Mayo, of Rochester, Minnesota, read a paper, entitled "Cholecystectomy with Special Reference to Removal of the Mucous Membrane of the Gall-Bladder in Certain Cases as a Substitute." This might be indicated in severe injuries or gangrene.

The importance of early operation for biliary calculi was advanced by M. H. Richardson, of Boston, Massachusetts. He believed that gall-stones should be removed as soon as the diagnosis was made. All his operations on the common duct were successful. In discussing this paper, Wyeth, of New York, endorsed the views of Richardson. Senn, of Chicago, believed that much might be learned by the X-ray. He did not operate in all cases, but only when life was in danger. In the latter opinion Bevan also concurred. In the dissecting-room he had found gall-stones in six-

teen per cent. of the subjects and in twenty-five per cent. of those over sixty years of age.

Dr. Evans presented for J. B. Murphy a paper on the "Present Status of the Murphy Button." Murphy believed that he had often been misquoted. In many cases operators had used imperfectly constructed buttons. A considerable amount of surgical knowledge was necessary to use this button properly. Seven hundred cases of gastro-enterostomy had been collected, with a mortality of nineteen per cent. Seven hundred and fifty cases of enterostomy, with nineteen per cent. Frequently the button might be passed by the patient unconsciously.

Dr. J. R. Pennington, of Chicago, called attention in a paper to the treatment of obstinate constipation, based on new points in the anatomy and histology of the rectum and colon. The tortuosity of the sigmoid, which does not always lie on the left side, with the formation of valves, might be a cause of constipation. For relief he had resorted to incision.

"Repair after Resection of the Intestine" was described by Dr. W. A. Evans, of Chicago. He had used the Murphy button, and there had been very little constriction.

Miles S. Porter, of Fort Wayne, Indiana, read a paper on "Appendicitis: Colitis as an Etiologic Factor: the Question of Removal of the Appendix in all Cases operated upon." He did not believe in removal of the appendix in all cases. Drainage was often sufficient.

John B. Deaver, of Philadelphia, read a paper on "Appendiceal Fistula." He considered this an important sequela of appendicitis, and spoke of certain cases in which the abscess had ruptured into neighboring cavities and structures. He believes in operating in appendicitis as soon as the diagnosis is made. In discussing these cases, Senn, of Chicago, stated that he had seen more cases of fistula following operation than when non-interference had been employed. It was going too far to advise an operation in all cases. Ordinarily the expectant plan of treatment would effect a cure in eighty per cent. of the cases. Dr. Mordecai Price endorsed the views of Deaver, as did Dawbarn and Mynter. Keen thought each case was a law unto itself, and did not approve of operating in all cases as soon as the fistula was diagnosed. He did not think one should wait for a second attack. Laplace be-

lieved that safety lay in an early operation. Ashton agreed with Deaver, as did Murphy.

The treatment of ventral hernia was considered by M. M. Johnson, of Hartford, Connecticut.

H. O. Marcy, of Boston, read a paper on "Inguinal Hernia in the Male." The essentials of a good operation are: careful dissection, proper disposition of the hernia contents, careful asepsis, and closure of the internal ring.

"External Drainage of Superficial Lung-Cavities, with Report of Two Successful Cases" was the title of a paper by W. L. Wills, of Los Angeles, California.

SECTION ON OBSTETRICS AND DISEASES OF WOMEN.

The chairman, W. E. B. Davis, of Birmingham, Alabama, reviewed the progress made in obstetrics and gynæcology during the past year. He believed that in this country much good would result if both obstetrics and gynæcology were combined. Fifty per cent. of the cases at first are properly obstetrical. He did not believe that the patient should be made to use a pessary when the safe operation could be done. In surgical operations rubber gloves should be worn.

J. H. Carsten, of Detroit, read a paper on the "Value of the Angiotribe in Vaginal Cœliotomy," in which he approved of conservative operations. The angiotribe was especially indicated in pus-tubes, ovarian cysts, and inflamed appendages.

Dr. Hugh M. Taylor, of Richmond, Virginia, read a paper on "Angiotripsy in Abdominal Surgery." He has used the angiotribe in twenty cases, and has had no bad results.

Dr. Henry P. Newman spoke on the improved technique in major and minor surgery of the female generative organs. He thinks much harm has been done by an improper use of the curette. If rapid divulsion is done, a tear may result. He has employed the angiotribe, and prefers the Doyen-Thumin instrument. J. Riddle Goffe, of New York, has used the angiotribe, and considers it perfectly safe. He prefers myomectomy *per vaginam* to hysterectomy.

Dr. H. J. Boldt, of New York, read a paper on "Myofibroma

Uteri." He believes that these tumors are pure myomata. They may disappear without treatment during the menopause.

Dr. John M. Duff, of Pittsburg, thought a laceration of the cervix should be repaired at once. Joseph Price, of Philadelphia, believes that there are few obstetricians at the present time. He deplored the use of the fountain syringe, which was too often used by all the members of the family.

A symposium on the relation of pelvic and intra-abdominal diseases to nervous diseases had been arranged. The first paper read, by Dr. Henry O. Marcy, of Boston, described "The Causal Relation Intra-Abdominal Diseases bear to Nervous Disturbances recognized by Gynæcologists, ignored by Neurologists." In making a diagnosis we should consider that nerves are both afferent and efferent. The wise gynæcologist should be a physician, a surgeon, and a neurologist.

"Neurosis due to Auto-Intoxication from Faulty Menstruation" was considered by Dr. Arthur Johnstone, of Cincinnati. In his opinion, every child-bearing woman has a surplus of nourishment which must be removed in some way. If not taken up by the child, it is thrown off by the emunctories. In young girls suffering from menstrual disturbances the symptoms are due to faulty elimination.

Dr. George H. Noble, of Atlanta, read a paper on "Traumatisms and Malformations of the Female Generative Apparatus as a Cause of Insanity." In reply to the question indicated by the title of the paper, which he had sent to alienists throughout the country, some gave a positive "No." Child-birth, sepsis, adhesions, and masturbation often acted as the cause. He had never seen a case of insanity or melancholia cured by an operation.

A paper, entitled "Gall-Stones and Diseases of the Gall-Bladder, and Nervous Symptoms resulting therefrom," was read by Edwin Rickets, of Cincinnati. He made a plea to operate in all cases of biliary calculi as soon as the diagnosis was made. In the discussion, Dr. Porter, of New York, stated that he often operated in cases of insanity and neurotic persons. Dr. Zinke, of Ohio, thought that every woman, before being committed to an insane asylum, should be subjected to a vaginal examination. J. M. Baldy does not believe that insanity results from pelvic trouble.

SECTION ON DISEASES OF CHILDREN.

The annual address was delivered by the President, Dr. Edwin Rosenthal, of Philadelphia, who spoke of the "Serum-Treatment of Diphtheria." Four thousand letters had been addressed to physicians during the past year, asking their views on this subject, and the replies showed a practical unanimity regarding its value. Statistics from health boards and hospitals in one hundred and fifty-seven cities give a mortality of thirty-eight per cent. without antitoxin, and fourteen per cent. when it was used.

Dr. Martin W. Barr, of Elwyn, Pennsylvania, gave the results of fifteen years' study of the "Etiology of Idiocy and Imbecility." The causes were heredity, malnutrition, and accident. In his series thirteen per cent. had a family history of imbecility and five per cent. of insanity. In his opinion, consanguinity is not a cause of insanity.

J. Madison Taylor, of Philadelphia, read a paper on "Study of the Circulation in the Feeble-Minded." The results were based on a series of nine hundred and thirty-five cases studied by F. S. Pearce and himself. In the majority of cases the vaso-motor system was deranged.

A. C. Cotton, of Chicago, spoke on "Infantile Cerebral Palsies." Twenty-five cases were reported. Of these eighteen were females. Eleven were diplegic cases, seven idiots; of eight hemiplegics, two were idiots.

One session of this Section was devoted to infant feeding and diseases of the digestive tract. A. L. Sherman, of New York, demonstrated a home milk modifier.

Dr. Louis Fischer read a paper on the "Milk-Supply and Control at the Kaiserin Friedrich Hospital, by Adolph Baginsky and Paul Sommerfeld, of Berlin." The cows from which the milk is obtained are not allowed to drink slop or eat potatoes, as both are likely to lead to diarrhœa. These animals are fed upon fodder. All of the animals are tested with tuberculin. Both the full milk and the mixed milk are sterilized in bottles for thirty minutes at a temperature of from 100° to 101° C. Pure milk does not agree with dyspeptic children; hence it should be diluted.

Dr. W. P. Northrup, of New York, read a paper on "Infant

Feeding: Accidents and Incidents." The modification of milk so as to meet individual cases imposes a great responsibility upon the physician. He gradually increases the amount of fat, sugar, and proteids, so that at the end of from eight to ten months full milk is taken. H. D. Chapin, of New York, has adopted the following plan. The milk is placed in a quart bottle until the cream rises. By means of a cylindrical ounce dipper on a long handle the contents of the bottle are then removed, beginning at the top. The first nine ounces contain twelve per cent. butter-fat, four per cent. proteids, and four per cent. sugar. The first fifteen ounces contain eight per cent. butter-fat and the same percentage of proteids and sugar. Dr. Victor C. Vaughan, of Ann Arbor, stated that sterilization of milk does not destroy any toxins which may be contained in it. A temperature of 356° F. often fails to do this.

Dr. C. F. Wahrer, of Fort Madison, Iowa, read a paper, entitled "Shall Children be Kept from Measles and Exanthemata usually Incident to Children?" Quarantine should be instituted against scarlet fever, diphtheria, small-pox, and whooping-cough. Measles was not so severe during childhood; hence it was not always best to keep children away from this disease. Several present dissented strongly from the latter view.

Henry Koplik, of New York, read a paper on "Rötheln: a Distinct Affection apart from Measles and Scarlatina, and its Differentiation from these Exanthemata." He believes that rötheln is a distinct affection, and can be distinguished from measles.

Jay F. Schamberg, of Philadelphia, read a paper on the "Clinical and Pathologic Study of the Rash of Scarlet Fever, with Special Reference to the Origin and Character of the Desquamation." In his opinion, too much stress is laid upon desquamation as a sign in this disease. The rash consists of puncta, vesicles, and "goose-flesh" papules.

Annie S. Daniel, of New York, gave a demonstration of a specimen of fetal ichthyosis, with complete pathological and bacteriological examinations.

A paper, entitled "Fetal and Infantile Typhoid," was read by John Lovett Morse, of Boston. It has been proved that the typhoid bacillus can pass through an abnormal, and possibly a normal, placenta and infect the fœtus. This being so, the disease is essentially a septicæmia.

Dr. J. C. Wilson, of Philadelphia, recognized three types of typhoid: the fetal, the infantile, and the adult forms. The serum reaction should be employed to determine the diagnosis.

Dr. Louis Fischer, of New York, read a paper on "The Differential Diagnosis between Abdominal Typhoid and Appendicitis by Means of Iodine Reaction; Report of a Case." The solution used was Ehrlich's iodine gum solution, made as follows: sublimed iodine, one part; potassium iodide, three parts; distilled water, two hundred parts. A drop of blood is collected on the cover-slip and pressed as thin as possible and allowed to dry in the air. A drop of the reagent is now added and the specimen is immediately examined. If the blood is normal, the red corpuscles stain a dark yellowish color, the leucocytes a lighter color, and their nuclei lemon-colored. Small extra-cellular glycogenic bodies are also seen. If suppuration be present in the body, the extracellular glycogenic bodies are increased in number, the leucocytes assume a brownish hue, and often a dark reddish-brown. The reaction affects the polymorphonuclear neutrophiles only, never the eosinophiles.

Dr. Frederick A. Packard, of Philadelphia, read a paper on "Valvular Heart Disease in Children." In three of the cases reported no history of infection could be traced. Of the others, rheumatism had been present in thirty-four, chorea in eighteen, scarlet fever in eleven, and cerebro-spinal fever in two.

"School Break-Downs" was treated of by J. Henry Bartlett, of Philadelphia. Sixteen thousand children in five of the larger cities had been compelled to leave school, the principal causes assigned being hasty eating, lack of exercise, improper sleep, and too many examinations.

A. Campbell White, of New York, gave a demonstration of liquid air. It may be used as a spray or cautery. Liquid air does not kill germs, but suspends their activity. Anæsthesia from the employment of this reagent lasts for several minutes. To abort boils the spray is applied, the skin being pricked in various places. It is valuable in the treatment of carbuncles.

The secretary for Dr. Joseph Trumpp, of Munich, read a paper, entitled "Intubation in Private Practice, and its Perfection." Statistics from five thousand four hundred and seventy intubations done by fifty-five operators in Europe and America

show a recovery-rate of thirty-five per cent. previous to antitoxin days, and since its introduction the recovery-rate has risen to eighty-two per cent .

Dr. Alexander Klein read for Dr. James Bokay, of Budapest, a paper, entitled " Injuries due to Intubation." In twelve thousand cases there were only four which resulted in a false passage.

Dr. William H. Welch, of Philadelphia, said the safest place for intubated patients was the hospital. He did not think the favorable statistics regarding antitoxin were always reliable or fair. The mortality in the intubated cases in the Municipal Hospital of Philadelphia, where antitoxin is employed, is fifty-eight per cent. He prefers the metal tube, while Fischer preferred the rubber tube.

INDEX TO VOLUME II.

(TENTH SERIES.)

Bovinine

IS strictly a physician's preparation, and is introduced to the profession only. It is not a substitute for any medication, but a powerful auxiliary to it. It is most palatable and it can be given in any vehicle not incompatible with a preparation so rich in albumen. Given in small doses at first, it is readily retained by the most delicate or irritable stomach, and is of especial value in acute or chronic gastric disturbances, and intestinal disorders. It is an IDEAL invalid food, and is suited to all ages and all conditions. We do not wish to send samples and literature where they are not desired, but thousands of applications prove the desire of medical men to be frequently reminded of Bovinine.

A postal will bring you our scientific treatise giving clinical cases, and details of treatment for all cases.

THE BOVININE CO.,
75 West Houston St., New York.

LEEMING MILES & CO., MONTREAL. Sole Agents for the Dominion of Canada.